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CYCLOPÆDIA

OF THE

DISEASES OF CHILDREN

MEDICAL AND SURGICAL.

THE ARTICLES WRITTEN ESPECIALLY FOR THE WORK BY
AMERICAN, BRITISH, AND CANADIAN AUTHORS.

EDITED BY
JOHN M. KEATING, M.D.

VOL. II.

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CYCLOPÆDIA
OF THE
DISEASES OF CHILDREN.

PART I.
DISEASES OF THE SKIN.

DISORDERS OF THE GLANDS.

By JAMES NEVINS HYDE, M.D.

THE SEBACEOUS GLANDS.

SEBORRHŒA.

Derivation.—Latin *sebum*, "suet," and Greek ῥεῖν, to "flow."

Synonymes.—Latin, Acne sebacea, Steatorrhœa, Seborrhagia; French, Acne sébacée; German, Schmierfluss.

Definition.—Seborrhœa is a disease of the sebaceous glands, characterized by a quantitative or qualitative change in their secretion, which may then be discharged upon the surface as an oily fluid, or in the form of semisolid, fatty scales or plates, occasionally accompanied by dilatation of the orifices of the excretory ducts of the glands.

Etiology.—Seborrhœa may be due to anæmia, cachexia, perversion of the physiological function of the sebaceous glands as a consequence of causes operating upon the surface of the body, derangements of the alimentary canal, the infectious granulomata (tuberculosis, syphilis), the exanthemata, inherited tendencies, and neglect of the rules of hygiene.

Pathology and Pathological Anatomy.—Seborrhœa is essentially a functional disorder, without primary structural changes, of the sebaceous glands. In some cases, however, where the disease has existed long unchecked, the glands may undergo atrophic changes; at least the epithelium lining the crypts of the glands becomes incapable of furnishing longer a catarrhal discharge. The secretion of a seborrhœa, examined microscopically, consists in varying proportions of free oil-globules and fat, epithelial cells, and amorphous granular masses.

Symptoms.—Seborrhœa may be of the oily form (seborrhœa oleosa), in which a fluid and oily secretion is poured out upon the surface; or of the dry form (seborrhœa sicca), in which the secretion is furnished in the form of fatty plates or scales. The disease may be general, involving the entire surface of the body. This is a rare and dangerous disorder, apparently allied to ichthyosis, in which, after the removal of the physiological vernix caseosa of the infant, the skin beneath is seen to be deep red in color, with a tendency to become fissured and to furnish rapidly a horny incrustation. Partial or local seborrhœa usually affects the scalp, furnishing thus a sequel to the condition represented by the pre-natal cap of vernix here accumulated. In this condition, thin or bulky, friable, greasy crusts of a dirty yellowish or brownish hue cover a slightly macerated, often ill-smelling surface. These may persist for months and lay the foundation for a future eczema of the region. In most severe pustular eczemas of the scalp in children the resulting crusts are in part built up of inspissated sebaceous matter furnished by a catarrh of these glands. In the dry form of seborrhœa of the scalp, whether symmetrical or affecting only portions of that region, fine grayish or yellowish scales accumulate, often pasting down the hairs to the surface of the non-inflamed scalp. In other cases they are freely shed from the surface. The disease when persisting may induce thinning of the hairs of the vertex at the time when puberty is reached. Seborrhœa of the face in children near the puberal epoch may form a greasy film of dirty yellowish-green, somewhat adherent crusts over the forehead, cheeks, or nose, beneath which the skin is inactive, and macerated or inflamed. In the latter case there are decided sensations of itching. Seborrhœa of the umbilicus in children is remarkable for the fetid odor of the secretion furnished, and for the reddened ring of eczematous skin surrounding the navel which usually complicates the disorder. In the genital region the tight prepuce of male children may imprison a fluid furnished by the sebaceous glands producing both local and, by reflection, general symptoms of disorder. The same local symptoms (fetid secretion, pruritic sensations, etc.) may result from accumulation of the secretion about the labia and clitoris of young girls.

Diagnosis.—Seborrhœa is distinguished from eczema by the abundance and fatty character of the oily secretion and of its scales and crusts, by the absence of the itching so characteristic of the latter disease, and by the absence of all inflammatory symptoms in the part affected. In psoriasis there are a more distinct definition, a more markedly circular outline, and more lustrous scales, the surface beneath them being reddened and exuding drops of blood when these scales are removed. In ringworm the trichophyton may readily be distinguished by the microscope. Lupus erythematosus of the face is characterized by darker patches and more adherent scales, which are never fatty, the patches moreover being more infiltrated.

Prognosis.—The prognosis of seborrhœa in children is decidedly favorable.

Treatment.—Internal treatment of this affection often requires at the outset an alterative cathartic, such as calomel or the gray powder, to be repeated as desired. The ferruginous tonics and cod-liver oil are indicated in many cases. The diet is to be regulated with especial care (excluding pastry, confectionery, hot bread, and oatmeal). The general surface of the body should be cleansed daily by a soap-and-water bath. Often the sulphide of calcium may be administered with advantage, in doses of one-tenth of a grain (.0066) three or four times a day. Arsenic is rarely indicated in these cases. Locally, all crusts should be softened by maceration in some fatty substance (almond or olive oil, vaseline, cold cream, glycerin-and-water), and then removed by washing in hot water and common toilet-soap, or green soap, or by the use of the alkaline spirit of soap of Hebra (sapo viridis two parts, alcohol one part, filtered and flavored with the tincture of lavender). After this a sulphur salve, one to two drachms (4.–8.) of precipitated sulphur to the ounce (32.) of salve-basis (lanoline, benzoinated sebum), may be applied. One ounce each (32.) of precipitated sulphur, alcohol, glycerin, tincture of lavender, and rose-water may also be shaken up together and used as a lotion. Another lotion popularly employed for this purpose is one in which to an ounce (32.) of cologne-water are added half a drachm (2.) each of glycerin and castor oil, and five minims (0.33) of carbolic acid. One to two drachms (4.–8.) of the tincture of cantharides or of the tincture of nux vomica may be added instead to four ounces (128.) of the rectified spirit of wine. Mercurial salves are also useful for the same purposes,—the ammonio-chloride, or red oxide, in the strength of from five to ten grains (0.33–0.66) to the ounce (32.). Carbolated, borated, and salicylated spirit lotions, one part of each to one hundred of cologne-water, with five parts of glycerin, are valuable for local applications to prevent the recurrence of these troubles. The spirit lotions are to be preferred in the local management of seborrhœa of the genital regions.

COMEDO.

Derivation.—Latin *comedo*, a "spendthrift."

Synonymes.—Fleshworms; Skin-grub; Latin, Acne punctata; German, Mittesser; French, Acné ponctuée.

Definition.—Comedo is an accumulation of inspissated secretion in the efferent duct of a sebaceous gland, exhibited externally as a yellowish or whitish pin-head-sized elevation or depression of the dilated follicular orifice, with a yellowish, bluish, or blackish central point.

Etiology.—The causes of comedo are practically those of seborrhœa, but the former is more often encountered in children. Biesiadecki and Kaposi suppose that the impact of the young lanugo hair against the follicular wall opposite its axis may be the remote source of the lesion which is betrayed later in the disorder of the gland. In a small proportion of cases the accumulation of filth upon the surface of the skin may be responsible for the trouble. More commonly there is general torpor of the secreting glands of the skin, associated with either visceral inactivity, chloroanæmia, malnutrition, or systemic poisoning.

Pathology and Pathological Anatomy.—The softish, cylindrical plug which may be expressed from the follicular duct is made up of roundish and flattened epithelial cells, free fat, fine lanugo hairs, cholesterine in crystals, pigment-granules (which furnish the dark color of the exposed extremity of the plug), and the worm-like, jointed, six-legged Demodex folliculorum (first recognized by Simon and Henle in 1841–42), which has no etiological significance in this case. Comedones have been found in children as early as the second year of life. In consequence of the pressure produced by the plug, the secreting walls of the sebaceous glands lose their characteristic structure. The lining epithelium of the dilated pouch below ceases to undergo the fatty metamorphosis requisite for the production of the secretion. This process, if long continued, results in atrophy.

Symptoms.—Comedones are present in almost every face, being conspicuous only when numerous. They are scanty and widely distributed, or numerous and closely packed, bluish or blackish, pin-head-sized points, observed usually in greasy-looking skins, often associated with lesions of acne, occurring rather rarely on the scalp, much oftener on the face, inside of the ear, neck, back, breast, and genital regions of the youth of both sexes, those especially near the puberal epoch. They are said to be rather more frequently encountered in blond males. When expressed, a yellowish-white, worm-like, cylindrical mass, with a conspicuous blackish head, emerges from the slightly-elevated, whitish rim of the follicle, from which circumstance is derived the vulgar name of the malady,—namely, the "black-head" or the "skin-worm." Scarcely any subjective sensation is produced. Crocker[1] calls attention to the frequency of comedones in children with a tendency to grouping and to development in the parts subject to heat and moisture. Cauty[2] reports the case of a boy ten years old, covered with short bristles one hundred to the square inch, proving on examination to be comedones. Cases of a similar sort have been reported by other observers.

Diagnosis.—The comedo should not be confounded with the blackish point produced by tar applied to the surface for medicinal purposes, or by alternate applications of mercury and sulphur resulting in a deposit of the black sulphuret of mercury on the skin.

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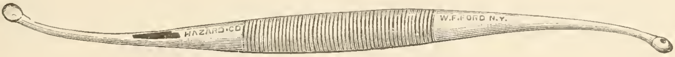
[1] Lancet, April 9, 1884.

[2] Med. and Surg. Jour., March 4, 1882.

Prognosis.—This is always favorable. In time even the most obstinate of comedones are exfoliated from the surface by physiological processes.

Treatment.—The comedo is readily expressed by the comedo-extractor (see illustration), after which the gland which has been constipated requires the treatment in general which is needful for the relief of seborrhœa. The affected part is to be washed in hot water, with or without the tincture of green soap and cologne-water, or the Sarg fluid soap (which is preferable for young children), followed by the application of sulphur salves,—one-half drachm to two drachms of sulphur to the ounce of salve-basis (2.–8. to 32.). Spirit lotions are also useful. After the bath, friction of the surface with a bit of white flannel on the finger is generally efficacious. A simple and elegant lotion for this purpose may be made by using half a drachm (2.) each of the tincture of benzoin and glycerin, to four ounces (128.) of rose-water. Weak solutions of corrosive sublimate, one-half grain to one grain (0.033–0.066) to the ounce (32.), may also be applied for the same purpose.



Piffard's modification of Unna's Comedo Extractor.

ACNE.

Derivation.—Greek ἀκμὴ, a "point."

Synonymes.—Stone-pock, Whelk, Pimples; Latin, Acne vulgaris, Acne disseminata; French, Acné boutonneuse; German, Finnen.

Definition.—Acne is a chronic inflammatory affection of the sebaceous glands and periglandular tissues, in which variously-developed papules or pustules, tubercles, or reddish blotches appear, usually upon the face or back, without producing marked subjective sensation.

Etiology.—Acne in its simpler forms is usually encountered at about the puberal epoch. It is not rarely seen earlier in life. It occurs in both sexes. It may be dependent upon gastro-intestinal derangements, anæmia, cachexia, accumulation of filth upon the surface of the body, struma, tuberculosis, and ingested medicaments.

Pathology and Pathological Anatomy.—The disease is usually caused primarily by constipation of the sebaceous glands, resulting rarely in folliculitis and peri-folliculitis, and possibly eventuating in the destruction of the gland and hair-follicle. A young acne-pustule examined in section usually exhibits evidence of vascular dilatation, exudation, infiltration of the walls of the acini of the glands, and out-wandered leucocytes. Still later, the parts are infiltrated with pus. The hair-follicle may be spared.

Symptoms.—In acne, reddish or violaceous, pin-head- to pea-sized, inflammatory papules, papulo-pustules, roundish or acuminate pustules and tubercles, few or numerous, often symmetrically disposed, appear upon the face (brow, nose, cheek, chin), the neck, or the back, often commingled and interspersed with comedones and minute roundish abscesses. Seldom there is produced a sensation of pruritus or burning,—occasionally the latter. Often a different course is pursued by individual lesions. Minute, slightly painful and tender papules may become pustular at the apex, indurated at the base and periphery, and a minute abscess result which bursts, crusts, and is followed by a reddish blotch, quite rarely by the formation of a punctate scar. In children the neighboring lymphatic glands become at times tumid and tender. Acne punctata is characterized by the development of papules, with a whitish or blackish comedo-centre. Acne papulosa, pustulosa, indurata, atrophica, and hypertrophica, are terms describing respectively papular lesions, pustular lesions, lesions having engorged and indurated bases, and those leaving cicatriform or hypertrophic relics of the inflammatory process.

Acne cachecticorum occurs in strumous patients, the lesions being small, violaceous papulo-pustules, which may be generally displayed on the body.

The most common form of acne in children .is acne medicamentosa, developing as a result of the ingestion of the salts of bromine and iodine. In these cases reddish and purplish, conical and roundish papulo-pustules, with marked inflammatory bases, patches of infiltration, abscesses, and even carbuncular lesions leaving scars, may result solely from the medication described.

Diagnosis.—Syphilis is readily distinguished from acne by the localization of the lesions of the latter disease, and by the concomitant symptoms of the former malady. In any given patient, as well as in other children of the same family, the absence of signs of congenital disease is important. Acquired syphilis of children is very rare. Syphilitic papulo-pustules of the face tend to cluster about the angles of the lips. The scalp, anus, and other regions of the body usually furnish evidence of any specific disorder present. Acne rosacea is not seen in children. Variola is an acute exanthematous disorder with vesico-pustules characteristically umbilicated. Impetigo and impetigo contagiosa have characteristic bulky crusts. Acne is symptomatically not a disease of such type. Its crusts are always an insignificant part of the symptoms present.

Treatment.—The internal treatment of acne is largely that indicated by the general condition of the patient, including the correcting of gastro-intestinal disorders, the use of ferruginous tonics when indicated by anæmia, and cod-liver oil when nutrition is impaired. Occasionally glycerin may be administered with advantage, in teaspoonful doses twice daily; or calx sulphurata, in doses of one-tenth of a grain (0.0066) three times a day; or, in place of the latter, the sulphide of lime. Arsenic is not required for children thus affected. The bowels should be evacuated daily and injurious articles of food carefully eliminated from the dietary,—for example, oatmeal, cracked wheat, and wheaten grits, the smaller seed-containing berries, hot bread and cakes, pastry and confectionery. Popular prejudice to the contrary notwithstanding, fresh meats need not be excluded. Regular intervals should be observed between meals, and no food should be taken during these intervals. The entire body should be scrubbed daily from head to foot in cool water, in order to stimulate the secretory apparatus.

Locally the affected parts may be well shampooed with either the alkaline spirit of soap of Hebra, already described, or the Sarg fluid soap, or the Rieger soap, with hot water. After this a sulphur salve may be applied, as in the management of seborrhœa. While the shampooing is in progress, all pustules should be opened with a fine, thoroughly-disinfected needle in a needle-holder, and the purulent contents expressed. In place of the shampoo, a lotion may be applied containing one or two drachms (4.–8.) of the tincture of benzoin and glycerin to the ounce (32.) of cologne-water; or some modification of Kummerfeld's lotion,—*e.g.*, precipitated sulphur, two drachms (8.); powdered camphor, two grains (0.133); powdered gum tragacanth, ten grains (0.66); lime-water and rose-water, of each half an ounce (16.). Van Harlingen employs one drachm (4.) of the sulphuret of potassium and the sulphate of zinc to four ounces (128.) of rose-water, with a similar purpose in view. Weak lotions of corrosive sublimate, from one-eighth to one-half of a grain to the ounce of spirit (0.008–0.033 to 32.), may be employed with advantage. Salves may also be used containing from five to ten grains (0.33–0.66) of either the ammonio-chloride or the yellow sulphuret of mercury to the ounce (32.) of salve-basis. A simple and ready method of local treatment in the case of children is the rubbing into the skin once in the day of finely-powdered sulphur scented with the oil of roses. This is best applied in the evening, after which it may be left on the surface during the night.

Prof. Unna, of Hamburg, has lately advised for external use, in the evening before retiring to bed,—

℞ Benzoinated zinc ointment, 86 parts;
Precipitated sulphur (or resorcin), 10 parts;
Silicious earth, 4 parts;

and by day,—

℞ Resorcin, 2 to 5 parts;
Glycerin, 1 part;
Orange-flower water, 20 parts;
Alcohol, 80 parts;

or

℞ Corrosive sublimate, 0.05 to 0.2 part;
Glycerin, 1 part;
Orange-flower water, 20 parts;
Alcohol, 80 parts.

MILIUM.

Derivation.—Latin *milium*, a "millet-seed."

Synonymes.—Latin, Grutum, Strophulus albidus, Acne miliaris; German, Der Gries.

Definition.—Milia are firm, isolated, pin-point- to split-pea-sized, hemispherical bodies, having a pearly lustre, covered only with epidermis, embedded within and usually projecting slightly above the general surface of the skin.

Etiology.—Milia are produced by congenital, traumatic, irritative, or inflammatory occlusion of the efferent duct of one or more acini of a sebaceous gland. The stricture of the duct producing this lesion has followed attacks of erysipelas, pemphigus, and mere erosions of the surface of the skin. The lesions themselves represent an accumulation of secretion behind the point where the excretory duct of the gland has been occluded.

Pathology and Pathological Anatomy.—Milia contain a mass made up of sebum, altered epithelium, fat, and small hairs, often mixed with a yellowish fluid encapsulated within concentric layers of fibro-cellular membrane, which also divide the milium-lesion into septa. There are no signs of resulting inflammation. The layers of epidermis above the mass of the milium are unaltered. Dr. Robinson, of New York, believes that milia result from misplaced embryonic epithelium carried away from a hair-follicle.

Symptoms.—Milia are firm, pin-head- to pea-sized, single or numerous, whitish, roundish or semiglobular bodies of a peculiar pearly lustre. They are often seen partly embedded in the skin over the temples, near the eyes, or about the cheeks, nipples, and genital regions of the young of both sexes. They are frequently seen on the faces of infants at birth, though often also encountered in adults. They are not the source of subjective sensation. After persisting for an indefinite period of time, they may be exfoliated by physiological processes.

Diagnosis.—In comedo, the black head of the plug and the distinctly-distended orifice of the duct exhibit striking differences from the smooth, shining, homogeneous surface of a milium, which, further, is found in more healthy skins than is the former. Sudamina, which resemble milia in appearance only, are easily recognized by puncture, which releases translucent fluid contents; xanthomata are yellow in color, and cannot be turned out from the skin in which they are lodged by mere efforts at expression.

Prognosis.—Milia, when untreated, are usually in time thrown off from the surface of the skin with its natural exuvium.

Treatment.—Milia may be removed by erasion with the dermal curette, incision, and subsequent application of a caustic (a crayon of the nitrate of silver, nitric acid, etc.). The simplest and most elegant, however, of all the methods is by electrolysis, the lesions being punctured with a fine needle in an insulated needle-holder connected with the negative pole of from two to four cells of a galvanic battery, the positive pole being connected with a sponge moistened with salt-and-water and held in contact with the patient's skin. There is scarcely any scar resulting from this simple operation.

MOLLUSCUM EPITHELIALE.

Derivation.—Latin *molluscus*, "soft."

Synonymes.—Acne varioliformis, Molluscum sessile, Condyloma subcutaneum, Epithelioma molluscum.

Definition.—Epithelial mollusca are smooth, roundish, hemispherical, often flattish, pin-head- to bean-sized and larger, whitish and waxy-looking or pinkish bodies, situated either upon or within the skin, often with a central or lateral point of depression, which resemble warts.

History.—The disease was first described by Bateman in the year 1817, under the title molluscum contagiosum. It has since been termed molluscum sebaceum. It is, however, no longer counted as a disorder of the sebaceous glands.

Etiology.—The question of the contagiousness of mollusca is still unsettled, with authorities on both sides. Eczema, profuse diaphoresis, lactation, and maceration of the skin are said to predispose to their occurrence. No other causes are cited. The questions of etiology and contagiousness are nearly the same as in the case of ordinary verruca, or simple warts.

Pathology and Pathological Anatomy.—Upon section, mollusca are found to contain either a semi-fluid, cheesy substance, or smooth oval bodies (molluscum-corpuscles), mingled with fat and epithelium. The body, as a whole, results from colloid metamorphosis of the prickle-cells lining the sebaceous glands and the root-sheaths of the hair-follicles. This change also affects the portions of the rete penetrating between the papillæ of the corium.

Symptoms.—Epithelial mollusca are firm, roundish, semiglobular, waxy whitish or rosy, pin-head- to cherry-sized bodies, sessile or pedunculated, which exhibit a whitish or darkish point centrally or laterally situated, resembling a comedo. Though rare, they are not infrequently seen on the face (eyelids) and neck, on the penis and scrotum of the male, and on the labia of the female; as also over the back and extremities. They are most common in children. They somewhat resemble roundish pearl buttons, especially when they have a flattened or a depressed summit. They are very rarely grouped, and still more rarely attain the size of a cocoanut. They develop slowly, and may be the seat of a mild grade of inflammation.

Occasionally, when they are ruptured, milky fluid contents may be expressed from these lesions.

Diagnosis.—Fibromatous mollusca, as they are often called (more properly fibromata), resemble these bodies in name only. In their firm connective tissue, their history, career, and external appearance, they are wholly different from the lesions here described. Mollusca may be distinguished from ordinary warts by their rounded shape and by the depressed point resembling a comedo. Small pigmentary nævi are readily recognized by their color, and often also by the hairs which spring from them.

Prognosis.—Mollusca are readily removed by treatment, after which they do not recur. When neglected, they commonly disappear by physiological exfoliation.

Treatment.—Mollusca can be removed by erasion, by ligature, by electrolysis, exactly in the manner described in considering the treatment of milia, or by the scalpel or scissors, followed by cauterization. Stimulating frictions, with green soap, and the application of white precipitate and sulphur salves,—the first in the strength of a scruple (1.33) and the last-named in that of a drachm (4.) to the ounce (32.),—also prove effective.

ASTEATOSIS.

Derivation.—Greek *α* privative, and *στέαρ*, "fat."

Synonymes.—Xeroderma, of Wilson, Asperitudo cutis.

Definition.—Asteatosis is characterized by a general or partial congenital absence or acquired diminution of the sebaceous secretion of the skin.

Etiology.—The disease may be congenital or produced by malnutrition, cachexia, disorders of the nervous system, or other cutaneous affections.

Pathology and Pathological Anatomy.—The skin, when examined, is found to be destitute of its normal sebaceous unguent. There may be absence, atrophy, or temporary suspension of function merely, of the sebaceous glands. As a result, the horny layer of the skin often becomes heaped up at various points of the surface, so as to produce a species of cutaneous keratosis.

Symptoms.—In asteatosis the skin is dry, inelastic, less extensible than normal, and destitute of its usual objective unctuous feeling. The hairs are usually thinned, staring, and lustreless, or absent. The nails also may be rugous and friable. The skin, in consequence of these changes, often becomes fissured and oozing, or scaly and crusted, in the regions involved in these changes. The slightest grade of this disorder is betrayed in some of the febrile processes in childhood; the gravest, in severe ichthyosis, lepra, and inherited syphilis complicated with marasmus.

The congenital forms of this disorder, known as ichthyosis sebacea and ichthyosis testacea, are extreme manifestations of this condition, where the child is brought into the world wholly unable to seize the nipple on account of the condition of its lips. The general surface of the body is then usually represented by a horny and shell-like cuirass of epidermis.

The **diagnosis** is readily established from a consideration of the symptoms named above.

The **prognosis** is favorable only in mild cases; the grave cases are remediless. In the former, as a rule, the symptoms are much less conspicuous in the summer season than in winter; while in the latter cold climates are far more prejudicial to the condition of the skin.

Treatment is to be conducted by the external application of oils (almond, neat's-foot, suet, palm oil, vaseline), and in the case of viable children by their removal to a congenial climate.

THE SWEAT- OR COIL-GLANDS.

ANIDROSIS.

Derivation.—Greek α privative, and ὕδωρ, "water."

Synonymes.—Anidrosis, Hypohidrosis.

Definition.—Anidrosis is that morbid state of the skin in which there is a total absence or quantitative diminution of the sweat effused upon the surface.

Etiology.—This condition may be a symptom of acute febrile disorders, chronic skin-diseases, or affections of the nervous centres or nervous trunks, as well as of disorders of the viscera.

Pathology and Pathological Anatomy.—Partial anidrosis may result from obliteration of the sweat-pores or coil-glands, limited to certain areas of the skin, where inflammation, degenerating new-growths, cicatrices, and other pathological processes have either mechanically obliterated the glands or by nutritional changes starved them into atrophy. In general anidrosis there is merely a functional disorder of the perspiratory system, without structural changes in the glands themselves.

Symptoms.—The term anidrosis implies complete absence, or complete suppression, of the sudoral function. Hypohidrosis is the more common relative diminution in the quantity of the sweat-secretion. It is exceedingly rare, if it ever occurs, as an idiopathic disorder, but is a common symptom of a number of disorders of the skin and other organs, more particularly in the febrile state. When it exists in a partial or complete form, the skin is dry and distinctly destitute of its natural moisture. It may be hot and dry, as in a fever; or cool and dry, as in ichthyosis and the several neuroses. The skin is affected with anidrosis in infantile spinal paralysis, which may be taken as a type of the anidroses due to nervous disorders, but the sweating returns when the nutritional and motor activity of the limbs are restored. The affections of the sympathetic nervous system and the trophoneuroses in children are similarly betrayed in temporary or persistent anidrosis.

The **diagnosis** of this affection is readily established by recognizing the moistureless condition of the integument, and the existence of a disorder capable of producing such a symptom.

The **prognosis** and **treatment** are those of the disease of which the anidrosis is recognized as symptomatic.

HYPERIDROSIS.

Derivation.—Greek *ὕπερ*, "in excess," and *ὕδωρ*, "water."

Synonymes.—Idrosis, Hydrosis, Ephidrosis, Sudatoria, Polyidrosis, Hyperhidrosis.

Definition.—Hyperidrosis is an effusion of the sweat-secretion in relative excess, the fluid accumulating visibly upon the surface of the skin.

Etiology.—Hyperidrosis may be pathological or physiological in character. It is rarely congenital, more often acquired. It may be due to disorders of the nervous centres, or to systemic states (pyretic remission), or to disease of the circulatory system (heart and blood-vessels).

It may be due simply to elevated temperature (aided by excess of clothing, summer weather, or the air of an overheated apartment), to unusual exertion, or to ingested medicaments.

Pathology and Pathological Anatomy.—When examined in section, the coil-glands and sweat-pores are not recognized as having undergone changes in cases of profuse hyperidrosis. The disease is a purely functional disturbance of the apparatus designed for the secretion of sweat.

Symptoms.—Localized hyperidrosis is limited to certain definite regions, such as the hands, feet, axillæ, groins, temples, and genital regions. In generalized hyperidrosis the sweat is poured out in excess from all parts of the body. The rapidity and quantity of this transudation vary in different cases with the condition of the atmosphere and the circulation. Children, and particularly infants, are especially liable to physiological hyperidroses when kept in apartments where the temperature is unduly elevated, or when they are too warmly clothed. Often the sweat thus effused has an offensive odor, but this is much less frequently noticed in children than in adults. Rarely, however, this occurs also in the former class of patients. Erythema and intertrigo (of the groins, the back of the neck, the axillæ) are frequently thus induced, and may lay the foundation for a severe eczema of this region. Sudamina may result in the form of minute lesions resembling seed-pearls, which are filled with a droplet of sweat.

The **diagnosis** is readily established by considering the moist and sweating condition of the skin.

The **prognosis** is usually favorable,—the only grave conditions being those in which the excessive sweating is a symptom of a formidable disorder.

Treatment.—In general hyperidrosis due to adynamic states, the ferruginous tonics, mineral acids, and quinine are usually indicated. Many children require special attention to the digestive function, including a proper dietary, and hygienic regulation of the bodily clothing, the coverings of the crib or bed, and the temperature of the apartments in which they sleep and play. Children habitually overheated are in as much danger of disease as those whose surface is habitually chilled. Externally, bathing with water to which a small quantity of salt has been added, usually one-quarter of a pound to the gallon, or with soap-and-water, usually by sponging and followed by brisk friction of the surface, is valuable when not contra-indicated by any systemic or visceral disease (cardiac cyanosis, anæmia, etc.). As a rule, whether salt be employed in the bath or not, a good reaction should in every case be established by friction. When the circumstances are favorable, nothing surpasses in value sea-bathing in summer temperatures. The sweating surface after the bath may be dusted with talc, boric acid, rice-flour, lycopodium, or finely-powdered starch containing from three to five per cent. of salicylic acid. Spirit lotions may also be employed containing from one to two per cent. of quinine, alum, or salicylic, tannic, or carbolic acid. Tar should not be used for this purpose upon the skins of children; and salves are not required if there be no complication in the form of an erythema, an intertrigo, or an eczema. When indicated, the unguentum diachyli albi of Hebra (see Eczema), or benzoated zinc salve, may be employed, in the usual strength.

Sudamina are best treated by simply dusting them with a fine salicylated starch powder.

INFLAMMATIONS.

By W. A. HARDAWAY, M.D.

ERYTHEMA.[1]

Definition.—For the purposes of this article, simple erythema may be defined as a redness of the skin, that temporarily fades upon pressure, and that appears in the form of diffused or circumscribed, variously-sized lesions, usually without elevation above the integument. It must be acknowledged, however, for reasons that cannot be detailed here, that any hard-and-fast definition is difficult to make, and the one adopted is solely in the interests of clinical convenience, and not with any thought of securing pathological accuracy.

Symptoms.—So far as the local expression on the skin is concerned, the eruption may appear in the form of patchy redness, or in diffuse areas, or in streaks and stripes of different sizes and shapes. The older writers restricted the term erythema to lesions of the kind just described, but if the cutaneous congestion made its appearance in finger-nail-sized spots, or assumed various punctate, annular, and gyrate forms, it was called roseola, with a qualifying adjective indicating peculiarities of shape, etc. In what was called roseola infantilis, the patches of congestion were described as of small size, closely grouped, and in general appearance not unlike the eruption of measles. There is really no warrant for these distinctions, and there is no question that mild cases of scarlet fever, rubeola, and rötheln were responsible for much of the cumbersome and intricate divisions of former times.

Etiology.—The causes of simple erythema are numerous and of the most diverse character. It may be *idiopathic* or *symptomatic*.

IDIOPATHIC ERYTHEMA.

This form of erythema is brought about by the influence of external irritation upon the skin, which, if left unchecked, may go on to true inflammation.

Thus, among the numerous causes of this condition may be mentioned erythema from heat and cold (erythema caloricum); erythema from pressing, rubbing, scratching, and the congestion arising from ill-fitting garments, instruments, etc. (erythema traumaticum); the active disturbances set up by animal and vegetable poisons (erythema venenatum).

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[1] Owing to the manner in which the general subject of erythema has been allotted to different writers on this Cyclopædia, I have been obliged to follow a method of treatment not entirely in accord with my own views. I have therefore thought it best to adhere in the main to the prevailing system of classification, with the object of presenting definite clinical pictures, although sacrificing in some degree a desirable scientific exactness.

In a work on the diseases of children there are several varieties of idiopathic erythema worthy of more extended consideration, two of the principal conditions being chilblain and intertrigo.

Erythema Pernio.—Chilblains are localized erythematous congestions that are very common in weakly children, especially girls. The usual sites of the disorder are the feet and hands, generally the former, but it also may attack the nose, cheek, or ears. The disease begins in congestive patches from the size of a dime up to that of a dollar, which later may coalesce and form a continuous band. They itch, tingle, and burn most distressingly. After repeated attacks the affected skin may become covered with vesicles, which may break down, leaving an excoriated surface, that may ulcerate. Chilblains are liable to relapse each season, making their appearance in the fall and not disappearing till the advent of warm weather. The cause is to be found in vicissitudes of temperature. The habit of toasting the feet at the fire before going out into the cold, and immediately upon returning in-doors, is undoubtedly responsible for much of this suffering. It will be observed also that children thus affected are, as a rule, not in robust health.

Treatment.—The treatment is both internal and local. The prime object must be to give tone to the system. Of drugs the most useful is iron in some form, the bitter barks with the mineral acids, and in strumous subjects cod-liver oil and the hypophosphites, together with the lacto-phosphate of lime. Cold general sponging with brisk towelling is of great advantage. The habit of hanging over fires should be interdicted, and the child should be made to wear stout, easy-fitting boots and woollen stockings. It is recommended that the patient should sleep in a moderately warm room, and that knitted bed-slippers be kept on during the night.

For immediate relief, very hot water applications give the most comfort. A calamine-and-zinc lotion is very agreeable (zinci oxidi, ℥ss; pulv. calaminæ præp., ℈iv; glycerinæ, ʒi; liq. calcis, ℥vii). Painting the parts freely and frequently with the tincture of iodine is a method of great value. The linimentum belladonnæ (B. P.) is said to give great relief to the itching. When ulceration or sloughing occurs, the lesions must be treated on general surgical principles.

Erythema Intertrigo.—This form of erythema is always at first a simple hyperæmia of the skin, which occurs on parts of the body exposed to friction from the contact of opposed surfaces, and in children, especially, it is often evoked by the irritation of urinary and fæcal discharges. In severe cases the skin is hot and tender, there is a hypersecretion of sweat, the epidermis becomes macerated, and the parts are bathed in a muciform discharge, which frequently emits a highly offensive odor. Tilbury Fox stated that this discharge differed from that of eczema in that it did not stiffen linen. Under circumstances of neglect, the surfaces may become fissured, raw, and even extensively ulcerated.

The disease is usually found in the groins, the folds of the neck in fat babies, the gluteal furrows, the inner surfaces of the thighs, and the flexures of the joints. Intertrigo in infants may appear quite suddenly, and under proper management may last but a few hours; on the other hand, if neglected or improperly treated, it may persist for weeks. When it is symptomatic of internal disorders of a grave character, the course of the disease is considerably lengthened and persists in spite of the best-directed efforts at cure. It is most frequently encountered in hot weather, although in infants it may be observed at all times of the year.

Relapses are to be expected. According to Hutchinson, owing to the fatness of infants, the eruptions of syphilis occurring upon them are apt to take on the form of intertrigo, the irritation of the buttocks by fæces and urine inviting the syphilides to these situations.

Diagnosis.—There is little difficulty in the recognition of intertrigo occurring in infants; after adult life has been reached there are one or more affections with which it might be confounded.

The diagnosis from eczema is of no practical importance; indeed, at times the line of demarcation is difficult to appreciate. Intertrigo may closely resemble the erythematous syphilide; but, while the former is mainly limited to the buttocks and genital regions, the specific eruption may extend as far down as the heels; moreover, the color of the syphilide is significant, and other symptoms, such as mucous patches, etc., are generally to be discovered in the syphilitic child.

Treatment.—It is generally easy to prevent the occurrence of an intertrigo. Cleanliness is to be secured by ablutions with soft water and a bland soap, frequent change of diapers, the immediate removal of, and protection against, irritating discharges, and the use of a simple dusting powder (zinci oxidi, ʒii; pulv. sem. lycopodii, ʒvi). After the disease has become established, it is well to keep the parts separated by the interposition of pieces of lint, and to apply a powder that is somewhat astringent (thymol., gr. ss; pulv. zinci oleatis, ℥i). Duhring advises diluted lotio nigra in obstinate cases. In nearly all grades of intertrigo I have secured the most admirable results with Lassar's paste:

℞ Acidi salicylici, gr. x;
Zinci oxidi,
Amylis, āā ʒii;
Vaselini, ʒiv.
M.

I think a better formula is the modification of the above suggested to me some years ago by Dr. G. H. Fox:

℞ Acidi salicylici, gr. x;
Bismuthi subnitratis, ʒii;
Corn starch, ʒiss;
Ung. aq. rosæ ad ℥i.
M.

This paste should be spread thinly over the involved surface. It is not only directly curative in its effects, but also affords a most admirable protection from irritating discharges.

SYMPTOMATIC ERYTHEMA.

When we bear in mind the anatomical and physiological peculiarities of the skin, and the intimate connection of this organ with the system at large, it is quite comprehensible that many morbid states of the organism find local expression in circulatory derangements of the integument. One of the commonest of these disturbances is hyperæmia of various grades. Certain general diseases—*e.g.*, variola, diphtheria, cholera, meningitis, vaccinia, etc.,—are often preceded, accompanied, or followed by erythematous rashes. These need not be further mentioned here, as their description will be given in connection with those disorders in other sections of this work.

More or less temporary congestions of the skin are known to occur in consequence of the ingestion of various drugs (erythema medicamentosum), although it is true that these rashes usually represent true inflammatory processes.

One of the most frequent, and at the same time one of the most important in a negative way, of these symptomatic erythemata is the form commonly called erythema infantile, or roseola infantilis.

In the older works on dermatology, and in most books on children's diseases, many pages are devoted to a description of this eruption. An attentive reading of such descriptions will show, as already stated, that much of what is set down as significant of the so-called roseola really applies to a variety of other cutaneous diseases, more especially rötheln, mild cases of scarlatina and measles, and light attacks of urticaria. It is nevertheless true, as is well known to practical physicians, that evanescent congestions of the skin are quite common in young children who are teething, or suffering from some slight derangement of the alimentary canal. These rashes generally assume the roseolous form, and are accompanied by a slight elevation of temperature and perhaps some redness, without swelling, of the palate and fauces. It is said to be most common over the sacral region and buttocks. Its course is capricious, and it usually disappears in from a few hours to a few days without desquamation. The chief importance of this so-called roseola is from the stand-point of diagnosis. Its existence is often the cloak for ignorance and charlatanry. Much of what the laity and certain irregular practitioners call "scarlatina"—not knowing or conveniently ignoring the fact that scarlatina is the technical name for scarlet fever—is in reality this symptomatic erythema, which fact also explains the wonderful facility of its cure, and the statement that is often made that a certain person has had repeated attacks of the specific rashes.

If one bears fully in mind the essential characteristics of the acute exanthemata,—the heat of skin, the rapid pulse, the condition of the throat and tongue, the glandular engorgement, the location of the eruption, in scarlet fever; and the peculiar prodromal period, the general catarrhal symptoms, and the features of both the mucous and the cutaneous rash, in measles,—the difficulties of diagnosis will not be very great.

Rötheln, the mildest of the exanthemata, is not at all times diagnosticated with such facility, and the differentiation is occasionally far from easy. Rötheln, however, is manifestly due to contagion, several children of a family probably being attacked at the same time, the eruption is more like that of measles, the glands behind the neck are enlarged, and the eruption is of a more stable character. The fact that the true variolous eruption is often preceded by a preliminary erythema should also be borne in mind.

The skin of new-born children is marked by a discoloration, which is at first red, then becomes yellowish red, and finally, for a while, of a quite bright red.

There is still another form of erythema, which it is clinically convenient to mention here, that has been termed erythema papulosum of the new-born, or erythema neonatorum.[1] I have seen a number of examples of it, and its occurrence has occasionally given rise to much confusion in diagnosis. It makes its appearance in the first few days of life, and is thought to be due to the influence of external and unusual irritants acting upon the tender skin of an infant newly come into the world. The eruption consists of very minute red papules, seated upon a hyperæmic base, which can be made to lose their color upon pressure. The lesions are most pronounced upon the back and breast. They fade in a few days, and the most congested spots exhibit a slight desquamation. The mucous membranes are unaffected, and there is no evidence of systemic reaction.

The symptomatic passive hyperæmias, which may result from a variety of agencies, *e.g.*, heat, cold, mechanical causes, pathological states, etc., need no particular description here.

FURUNCLE.

Definition.—A furuncle is an acute circumscribed phlegmonous inflammation occurring round a skin gland or follicle, that terminates in suppuration, and the expulsion of a central slough, or core.

Etiology.—When boils occur singly it will often be found that they have been evoked by some local irritation, *e.g.*, the pressure of ill-fitting instruments, prolonged decubitus, or the teasing of the skin by a frayed or unusually rough garment.

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[1] A consideration of this disorder is hardly proper here, but I introduce it because it will probably be omitted from other sections of this work.

It is a thoroughly well recognized matter of experience that furuncles occur in connection with a variety of constitutional states of a depressing character; for example, in diabetes, after variola, measles, scarlatina, etc. It is also a common observation that certain local pruritic disorders of the skin are commonly accompanied, or more often followed, by boils. Thus, Von Rittershain states that after exfoliative dermatitis of infants they are very frequent; and the furunculosis that occurs as a sequel to eczema is very annoying and often protracted. Vogel declares that the children of tuberculous parents suffer much from furuncles at the occiput, and even over the whole head, which are accompanied by coincident swelling of the glands, and cause much suffering.

In the hot summers of this section of country children are very subject to prickly-heat, which is often accompanied by crops of furuncles. A most painful and persistent furunculosis is often seen in connection with the chronic intestinal catarrhs of children.

Children of some age are perhaps more liable to furuncles than infants, and it would seem that young boys are especially prone to them about the neck and back, at the same time being in no appreciable bad state of health. There is no ground for the opinion that a superabundance of good health predisposes to boils, as was formerly believed, although it is incontestable that physical well-being is no bar to their acquisition. Boils sometimes seem to occur epidemically, spreading through families and schools, and there is no doubt that pus from them is contagious, as was pointed out by the late Mr. Startin. I have recently seen a family, consisting of a mother and several children, who all suffered from boils, that were probably inoculated on them by flies from a neighboring slaughter-house. In the past few years the opinion has steadily gained ground that the furuncular inflammation is invariably due to micro-organisms that find entrance through the glands and follicles of the skin. Indeed, the conclusion has been reached by some investigators that chemical, mechanical, or thermic irritants, if entirely free from micro-organisms, cannot produce suppuration.

The presence of pus-cocci in the pus of furuncles, the clinical fact of contagion, and the successful inoculation of pure cultures would seem to establish beyond question the essentially parasitic nature of the process. The pus-cocci have been found in dish-water, the surface of the ground, and the wrappings of healthy sucklings; and Bockhart has cultivated them from scrapings of normal skin, from dirt under the nails, and from nasal mucus. As it is quite possible, therefore, that these organisms come in contact with, or are introduced into, our bodies without injury under ordinary circumstances, it follows that a favorable soil is necessary in order that they may exert a pathological influence,—such a soil, for example, as is found in general disorders of nutrition, in pruritic skin-diseases, etc.

Pathology and Pathological Anatomy.—Boils always begin around the hair-follicles and the sebaceous and sweat glands, and there is reason to believe, as stated above, that the inflammation is set up by the entrance of pus-cocci into these openings. According to the recent researches of Bockhart, the micro-organisms gain admittance either through the ducts of the sweat-glands or through the openings of the hair-follicles and sebaceous glands, or through abrasions or injuries to the skin. If they do not penetrate into the cutis, simple impetigo is the result; if they pass vaguely into the cutis through some lesion in the epidermis, a skin-abscess is the consequence; but if they pass along the duct of a sweat-gland or penetrate down the lumen of a hair-follicle, the process of suppuration is much more severe, and gives rise to the formation of a furuncle, of which the suppurating gland or duct forms the core.[1]

It is thought probable by some that the vessels surrounding a gland or follicle become blocked, the parts suffer necrosis, and the subsequent inflammation is set up around this tissue to get rid of it by suppuration (Crocker).

Symptomatology.—A boil may commence with a slight itching sensation, and presently there will be noticed a little pimple that is even at this time quite painful. Within perhaps twenty-four hours the lesion becomes more elevated, more tender, of a conical shape, and is surrounded a little later by a zone of reddened skin. At the apex of the swelling a point of suppuration is soon detected, and in a week or ten days the boil matures or becomes ripe. The pain, which at the beginning was of a pricking character, becomes a dull ache, accompanied by a constant throbbing and an uncomfortable feeling of tension. These sensations are apt to be increased in severity at night.

If pressure is made on a boil before it is mature, a little pus and blood will escape; but later, when the abscess bursts of itself, or is opened by the knife, the core becomes visible, although it does not, even at this time, come away with ease. So soon, however, as the core is extruded, the boil quickly heals, leaving in its wake a violaceous discoloration and after a while a minute cicatrix. Boils vary much in size; some are no larger than a coffee-bean, while others may be of the diameter of a silver quarter-dollar. Some boils also run their course more rapidly than others. When a core does not form, it is called a blind boil.

Furuncles may occur singly, or there may be present several at the same time; often, unfortunately, the morbid condition is kept up for weeks or months by a succession of crops. While there is generally very little constitutional reaction, when the so-called furuncular diathesis is established there may be, especially in children, great restlessness, loss of sleep, anorexia, and emaciation resulting from the constantly-recurring pain and free discharge of pus.

Boils may appear anywhere on the body with the exception of the palms and soles, but they have a special tendency to develop on the back of the trunk, and also frequently in children in the axillæ and along the borders of the lids (styes). They also may attack the ceruminous glands of the ear, in which situation they are excessively painful; here, however, they are not often seen in children.

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[1] Abstract in Medical Chronicle from Monatshcft f. prakt. Dermatologie, 1887, No. 10.

Diagnosis.—The diagnosis offers few difficulties. A boil may be distinguished from a carbuncle by its smaller size, its more pointed shape, and its single point of suppuration; whereas a carbuncle is generally solitary, is much flatter and larger than a boil, has an indurated border, and, in addition to its multiple openings, the overlying skin is completely destroyed, Boils should also be differentiated from the pustular syphilide and the eruption of ecthyma.

Prognosis.—The prognosis of boils is usually good. When, however, they occur in crops, even if the patient is otherwise well, they may prove very persistent and even appreciably depress the general health. When boils appear in connection with serious systemic disorders, their presence materially increases the sufferings of the patient.

Treatment.—Whatever view is taken of the furuncular process, it is the manifest duty of the physician to put his patient in the best possible condition of health. If it is thought that sewer-gas or arsenical wall-papers are causative factors, these should be removed and remedied. All local sources of irritation should be sought for and corrected. The dyspeptic, the anæmic, and the strumous should each receive appropriate treatment.

Very often change of scene and air is highly beneficial.

There are certain remedies that, given internally, are presumed to have some specific effect on boils.

Yeast is an old-fashioned "cure" that sometimes seems to exert a beneficial effect. I have never given it to children. An adult may take a half-wineglassful night and morning.

Bulkley extols the hypophosphite of sodium. The sulphide of calcium has been lauded in furunculosis. I have generally administered it to children in doses of one-tenth to one-fortieth of a grain four times a day. Although I have made use of this drug almost in a routine way for the last sixteen years, I am still unable to affirm positively that I have seen any constant or certain effect from it. It seems to me that at times suppuration is hastened in those taking it, but I have never known it to put a stop to the furunculosis. I have had far better success with the syrup of the hypophosphites, and in strumous children I have made much use of an emulsion of cod-liver oil, hypophosphites, pancreatin, and the syrup of the lactophosphate of lime. Le Gendre and Bouchard claim to have arrested furuncular eruptions by intestinal antisepsis.

It is well to endeavor to prevent or at least limit suppuration as much as possible; but when this cannot be accomplished it is advisable to hasten maturation and treat the abscess-cavity on antiseptic principles.

To secure the first object various methods have been suggested. Bidder, following Hueter, employs a two-per-cent. carbolic-acid solution with which he makes one or more injections according to the size of the boil. I should certainly not advise these injections for children. Theoretically, the method is excellent, but its practical execution is very painful and annoying. L. Heitzmann strongly recommends an eight-per-cent. salicylic-acid plaster or salve. Gingéot's favorite application is the tincture of iodine. It must be put on in successive layers and allowed to encroach a little on the healthy skin. He advises also that all other cutaneous lesions be similarly treated, to prevent their development into furuncles. Loewenberg makes use of a saturated solution of boracic acid. Verneuil advocates a two-per-cent. phenic-acid spray. The following application is recommended by Halle and Jamieson:

℞ Tinct. iodini, ℥i;
Acid. tannic., ʒss;
Pulv. acaciæ, ℥ss.
M.

Of late my own plan of treatment has been to apply to the furuncle a piece of Unna's carbolic-acid-and-mercury plaster mull, cut so as to cover the lesion and project a little beyond. Often this procedure will cause the boil to abort. On no account should poultices be made use of to encourage suppuration; they always do harm and seem to provoke new crops. Nothing succeeds so well in my experience as the Unna's plaster just mentioned in hastening suppuration where pus has already formed. After a few hours of its application it will generally be found that the boil has burst, or that the slightest prick with a knife or a needle will cause the pus to well out. A small hole may be cut in the centre of the plaster, corresponding to the apex of the boil. Squeezing and other manipulations should be avoided. After the furuncle has burst, the cavity should be dealt with antiseptically, the best agents being iodoform, iodol, or carbolized oil. Crocker says that sweat-gland boils are best treated by painting on a layer of collodion.

Although auditory furuncles are comparatively rare in children, when they do occur they cause much distress. I append the following notes on the treatment of boils in the ear, which my friend Dr. H. N. Spencer was kind enough to prepare for me:

"Treatment should have regard to the alleviation of pain, to resolution, and to prevention of the occurrence of others, to which there is liability. These indications are all met in the application of an ointment composed of extract of arnica, extract of belladonna, and morphine, and the use of compression. I have not known this treatment in a single instance to fail to procure speedy and permanent relief. The knife should *not* be employed; and poulticing, syringing, the instillation of warm water or drops of any character, are to be condemned, entering largely as they do as factors in the production of this form of ear-disease. The resilience that there is in absorbent cotton at the same time with its absorptive property constitutes it the best material out of which to make the compress.

"Pressure that is brought to bear uniformly upon all the walls of the canal prevents the development of furuncles by its influence upon the circulation, at the same time that it operates upon those which have formed to promote resolution or the culmination of their discharge.

"The after-treatment should look to the removal of the local cause, if this existed, in the form of inflammatory trouble, whether of the meatus or tympanic cavity. The yellow oxide of mercury in inunction is valuable as a means of stimulating the glands to renewed secretion."

PHLEGMON.—ULCERS.

By H. TUHOLSKE, M.D.

PHLEGMON.

Definition.—Phlegmon is an inflammation of the cellular or areolar tissue. This tissue is present in the human body from scalp to toe; subcutaneous, intermuscular, perilymphatic, perivascular, peritendinous, intrapelvic, retro-peritoneal, surrounding the structures of the neck and following them into the thorax; everywhere, as a bond of connection of the various tissues or organs. To describe the phlegmonous process of every locality and in its varying connections would be both interesting and instructive, but beyond the limits of this short article. In children, it has its foci of election in the neck and in the axilla, in the mammæ, on the forearm and hand, in the groin and on the buttocks, in the peri-anal and peri-cæcal spaces. It may be acute, diffused or circumscribed, chronic or malignant.

Etiology and Pathological Anatomy.—It is described as occurring idiopathically, but I believe it to be mostly secondary to an existing neighboring inflammatory or necrotic process, or of traumatico-septic origin. It is often associated with phlebitis or lymphangeitis, of which at one time it may be the cause and at another the effect; or with erysipelas, from which it differs in this,—that in phlegmon the cellular tissue is primarily inflamed, while in phlegmonous erysipelas the inflammation of the skin and cellular tissue results from the same cause, or the skin is affected first and the cellular tissue secondarily. It may be of puerperal origin, as described by Buhl. The pathological process is everywhere the same. The capillaries of the involved territory are dilated, and through their altered walls the blood-serum escapes into the tissues. Soon this exudate is augmented by the emigration, in large quantities, of the white blood-corpuscles; they increase rapidly and pre-existing connective-tissue cells wake into renewed activity. The presence of these young cells in vast numbers increases the tendency of the fatty and cellular tissue to undergo suppuration, and from their very pressure results necrosis of particles of adipose, or of the connective tissue, or of fascias. As soon as such necrosis has taken place, the suppurating process spreads in all directions, and, reaching the cutis, it succumbs likewise and allows the pus and detritus to escape. Generally this arrests the progress of the disease; the necrosed masses are removed; granulations fill the loss of substance, and cicatrization results. Phlebitis or lymphangeitis, septicæmia or hemorrhage, may complicate the later stages of this process.

Symptoms of Acute Phlegmon.—After the first day or two, when the patient complains of a tender, stiff, tingling feeling, the swollen part becomes shining and painful, frequently exquisitely so; the swelling is diffuse, uniform, slightly raised above the surface, and without a well-defined border. Although the skin does not participate primarily, it presents a reddish, erythematous appearance, which, as the disease progresses, becomes brawny, dusky, and œdematous. The swelling, which at first had been tough and inelastic, loses in firmness, becomes doughy and finally soft, and, if not too deeply situated, fluctuation becomes distinct. The suppurating process will now spread in the direction of least resistance, following the sheaths of tendons, which it involves, and along the veins and fascias, towards the integument, until this, in one or more places, eventually gives way and allows the discharge of pus and necrotic débris. As a rule, if nature has her sway, this takes place only after pieces of fascia have been destroyed, tendons have become necrotic, and the destructive process has spread far beyond its original limits. Then gradually the sloughs separate, a reparative process assisting in their removal, granulations form *en masse*, and the patient recovers,—some shortened tendon, contracted fascia, or fistulous tract remaining as lasting evidence of the destructive tendency of the disease. The systemic reaction is proportionate to the extent and intensity of the local process. The patient suffers with fever and with chills at the time of the pus-formation. In any case, thrombosis of involved veins may lead to infarcts in the lungs, or a thrombus, becoming septic, to suppurative embolic processes.

While pathologically identical, locality vastly influences the details of the course and termination of these cases. In the neck, according to the anatomical surroundings of the suppurating foci, pus may find its way out superficially, or burrow in the deep tissues or, following the reflections of the deep cervical fascia, into the mediastinal spaces. Beginning in the short connective-tissue fibres of the distal phalanx, necrosis of the primary focus results; inflammation follows the tendons, spreads through the palm of the hand, underneath the anterior annular ligament, up the forearm, and perhaps the arm. In the cone-shaped ischio-rectal space, filled with the low form of areolar tissue, the pus may discharge into the rectum through the perineum, and establish fistulæ and sinuses in and about the rectum, perineum, and buttocks.

Treatment.—While duly appreciating the value of general treatment and recommending to meet promptly every indication as it presents itself, I believe the local treatment to be paramount. Watch the patient's temperature and secretions, and administer, if the bowels are constipated, a laxative, and quinine and nutritious food. During the first days we may be able to assist in bringing about resolution. The remedies to be applied locally are mercurial inunctions, with absolute rest of the part and elastic compression, or, preferably, absorbent cotton wrung out of a warm two-per-cent. solution of carbolic acid, enough to envelop the affected part and cover far beyond it; cover this with oiled silk and retain it by a bandage snugly applied. This should be changed two or three times daily. Occasionally the hypodermatic injection of two minims of a three-per-cent. solution of carbolic acid—say three such to a square inch of involved territory—will be of value; or the emollient flaxseed and laudanum poultice may accomplish the object, and is undoubtedly more pleasant to the patient. Whenever the presence of pus can be demonstrated, incisions, deep and multiple, rather than extensive, in children, should be promptly made, and followed by thorough disinfection, complete drainage, and an antiseptic dressing according to the fancy of the operator. If the presence of pus cannot be demonstrated by the sign of fluctuation, because of its being too deeply situated, but is inferred from the œdema and pitting and intense localized tenderness, incision is demanded; in the distal phalanx (paronychia) incision to the bone, before pus is at all formed, is the best treatment.

The acute circumscribed variety of cellulitis is of frequent occurrence. It generally results in an abscess and in healing after the evacuation of the pus. The furuncle is a typical circumscribed phlegmon. An interesting variety is the abscess of the infant's breast, produced by the handling of the breast by ignorant and unclean nurses, the squeezing out of the milk, tight bandaging, etc.

The chronic form of cellulitis requires mercurial frictions, massage, elastic compression, and, as a rule, general tonic treatment.

The consideration of malignant cellulitis, due to septic traumatism, animal virus, or ptomaines, is beyond the purpose of this article.

ULCERS.

Definition.—An ulcer is a solution of continuity in the surface of the skin or mucous membrane, deeper than its epithelial covering, and maintained by causes local or general. In all cases it results from the molecular death of a portion of the skin or mucous membrane itself, a sequel to a suppurative inflammation, and disposed less to the formation of granulation-tissue than to a progressive destruction along its periphery.

Etiology.—The causes which on the one hand interfere with the formation of granulation-tissue, and on the other favor that progressive destruction, are so numerous that a simple classification of ulcers, etiologically, seems impossible. However, from the great variety of ulcers two large groups may be readily separated,—to wit, those depending upon an interference with the circulation or upon a prejudicial condition of the blood, and those resulting in the life-history of tumors: the remainder make up a group caused by mechanical violence, physical or chemical irritation, heat or cold.

Pathology and Pathological Anatomy.—It would almost appear out of place, in a short essay like this, to describe in detail the minute changes which the involved tissue causes before, by its removal, it leaves the ulcer. In its immediate periphery there results a dilatation of the blood-vessels, with a permeability of their walls, increase of nutritive juices in the tissues, emigration of the white blood-corpuscles, and finally a proliferation of pre-existing tissue-cells. This process separates the dead from the living tissues. The dead tissue, thrown off in bulk as slough, is the result of the process called gangrene; thrown off in minute molecular particles, it means ulceration.

With these particles is discharged a fluid, furnished by the inflammatory process,—pus, which is sometimes thin and flocculent, often grayish and bloody, and occasionally yellowish and creamy. The discharge from some ulcers is acrid and corrosive; from some contagious and, when rapidly decomposing, foul. Laudable pus, so called, is a sign of the ulcer's healing.

During the first or *spreading* stage of an ulcer its margin is hyperæmic and swollen, and, if it is spreading in the subcutaneous tissues more rapidly than in the skin, the edges are undermined. The base shows short-lived, dying granulations, has a grayish-yellow appearance, and is covered with pus and the débris of disintegrating tissue. As the *stationary* period of the ulcerative process is reached, the active inflammatory process, losing its intensity, becomes a process of repair. "The healing of an ulcer," says Paget, "differs in no material point from that of an open wound with loss of substance. It is a healing by granulations, and, though the shape and other characters of the cicatrix often have peculiarities indicative of the disease it has repaired, there is no known difference in the process of repair."

These general remarks are applicable to the mode of production and the pathology of all ulcers. The chief varieties of ulcers may be recognized by studying their deviations from the typical or simple ulcer. Now, what are its marks? It occurs in a healthy person, its base slightly depressed, uniformly covered with small, florid granulations, which feel soft, pliant, and elastic, and which, though highly vascular, do not readily bleed and are not painfully sensitive. The edges shelve gently down to its base, and feel scarcely harder. At their junction with the skin they are generally opaque and white, with a very slight thickening of the epidermis; within this they have a purplish-blue tint where the newly-formed epidermis veils the color of the healing granulations, and yet within this the granulations have a deeper hue than those nearer to the centre of the ulcer, being most vascular where the cuticle is most highly developed.

The pus from such an ulcer is healthy or laudable; the parts immediately beneath and around it are somewhat more vascular than is natural, but are not otherwise changed.

As departures from this type, depending mainly upon local conditions, we may find the pink granulations becoming intensely red, and dying; the border undergoing a rapid disintegrating process; the surrounding skin hot, swollen, painful, and œdematous; and the discharge thin, sanious, and sanguinolent. We designate such a one an *inflamed ulcer;* the granulations and borders may die off *en masse*,—a *sloughing ulcer;* the granulations may become pale, flabby, sodden with serum,—a *weak* or *œdematous ulcer;* the granulations may be florid, growing rapidly, bleeding readily, overlapping the edge, and highly sensitive,—an *exuberant* or *irritable ulcer;* or with an inflamed condition of the granulations there may be intense pain, or with no appreciable change in the granulations excessive sensibility and pain disproportionate to all the objective symptoms,—a *neuralgic* or *painful ulcer;* the granulations may become converted into a gray or grayish-yellow firm layer or rind,—a *croupous*, and, when attended with severe local inflammatory symptoms, a *diphtheritic ulcer*.

Numerous also are the deviations from the typical ulcer depending upon anatomical peculiarities, constitutional or specific diseases; and they express themselves by their peculiarities of shape, size, or color, by the contagiousness of their secretions, and by their behavior under the influence of medicinal agents. They might properly be called *symptomatic ulcers*. It would be foreign to this article to deal with *typhoid ulcers* of the intestine, which lie in the axis of the intestine and affect Peyer's patches and the solitary glands; *tubercular ulcers* of the intestine, which follow the course of the blood-vessels, the *dysenteric ulcer* of the rectum and colon, the *diphtheritic ulcer* of the throat, the *lupus ulcer* of the face, the *malignant ulcer* in any locality, the *chancroidal* or the *primary syphilitic ulcer* of the genitals, the *gouty* and *scorbutic ulcer*, ulcers due to *scabies*, and the gangrenous ulcers of vaccinia and varicella of Hutchinson. There remain as proper to this article the following forms:

VARICOSE ULCER, STRUMOUS OR TUBERCULAR ULCER, AND SYPHILITIC ULCER.

Varicose ulcers are such as are connected with an enlarged vein or a varicose state of the veins, and they are generally found upon the lower extremities. They rarely occur in children, except after an excessive formation of callus following fracture of the bones, after deep ulcerative processes of specific origin, or from the pressure of tumors connected with the bones or muscles. "They begin," says Pepper, "in one of four ways: first, by a rupture of the attenuated walls of a dilated vein; secondly, by thrombosis of a cutaneous vein and its capillary tributaries; thirdly, by an abrasion; and, fourthly, by the gradual transition of eczema to ulceration." The varicose ulcer is chronic in its character; its base studded with grayish-red granulations; generally depressed, with prominent edges; sluggish and indolent in appearance, surrounded by enlarged veins, and often by clusters of vesicles. It tends to perpetuate itself by increasing venous obstruction.

Strumous or *tubercular ulcers* furnish a large contingent to ulcerative processes in children. They result from strumous inflammation in the subcutaneous tissue or lymph-glands, but they may appear without such preceding diseases. They are most frequently found in the neck, under the jaw, or along the course of the glandulæ concatenatæ, in the axilla, in the groin, at the back of the knee, or upon the face. They are generally multiple, of irregular shape, approaching an oval, a number of them coalescing, with undermined edges which look pale or purplish; they secrete a thin, sanious, and often flocculent cheesy secretion; the skin in the neighborhood of the ulcer appears sometimes natural, more often purplish, and frequently it is undermined by fungous granulations, smooth, spongy, and friable, like those covering the base of the ulcer. It occurs in the class of patients generally called scrofulous,—a class of patients with thin, delicate skin, large lymphatic spaces rich in lymphatic net-works, where every irritation of the skin or mucous membrane finds ready response in an enlargement of neighboring lymphatic glands, resulting in a granular inflammation or caseation.

Syphilitic ulcers.—Owen, in his "Surgical Diseases of Children," cites an instance of a primary ulcer upon the prepuce of a boy nine years of age. This is so rare an occurrence that we may safely say that syphilitic ulcerations in children are most frequently due to broken-down gummata, tubercular syphilides, mucous patches, or condylomata. Inherited syphilis is by far the most frequent cause of syphilitic ulceration in children, and in the infant of ulcerative condylomata about the anus, between the nates, in the angles of the mouth, and in the mucous membrane of the nose. Deep ulcerations occur after the lapse of a few years. The gummatous ulcer of inherited syphilis differs in nothing from the gumma of tertiary (acquired) syphilis. It follows a swelling originally connected with the bone or periosteum, or in or beneath the skin or mucous membrane. It may grow quietly like a cold abscess, and may open spontaneously. Its edges are irregular and abrupt, the base raised, covered with tissue gray or grayish-pink in color, of glistening appearance and firm consistence; if pierced by a probe it is found to be much firmer than ordinary granulation-tissue, to bleed but little, and to be insensitive; it is very enduring, and is quite distinct from slough, leaving a deep, first brawny, finally white cicatrix. It may cause deep destruction of tissue.

Diagnosis.—No diagnosis of an ulcer is complete unless it includes a recognition of its condition,—the character of its base, its granulations, its edge, its surroundings, its secretion, the presence or absence of pain, and finally its cause. The base of an ulcer may be shallow or deep, showing the extent of loss of substance; it may be irregular or ragged, showing that it is spreading; glistening and smooth, indicating want of action; or it may be covered by a thick, grayish membrane, like the croupous. The granulations may be small, bright red, and uniform in size, neither bleeding readily nor excessively painful, showing a favorable progress; or intensely red, florid, and of unstable existence, showing its inflammatory condition;

or pale, soft, and œdematous, showing its weak vascularization; or large, spongy, and exuberant, overreaching its base, as in a burn; or fungous, as in a scrofulous ulcer; or they may have the characters described as belonging to a diphtheritic or syphilitic condition. The edge of an ulcer may be smooth, gently sloping, soft, and pink-colored, as in a healthy ulcer; or hard, prominent, rigid, and adherent, as in an indolent ulcer; irregular, undermined, and purplish, as in a strumous, or irregular, ragged, and breaking down, as in an inflamed or sloughing ulcer. The skin around its periphery may be normal; or eczematous, as in a varicose ulcer; red, angry, and hot, as around an inflamed, or slightly purplish and cool, as around a strumous ulcer. Its secretion may be small in quantity, yellowish, and creamy, as in a healing ulcer; thin, sanious, flocculent, as in a strumous; abundant and thin, showing a rapid breaking down of granulation-tissue. It may show an excess of lime salts, indicating its connection with bone. It may contain pathogenic micro-organisms, showing its septic or, it may be, its infective nature. Its pain may be barely noticeable, or it may be stinging and burning, as in an inflamed ulcer, or lancinating and severe, as in an irritable, painful, or neuralgic ulcer.

Ulcers may show in their multiplicity the fact of a constitutional origin; by their locality, their specificity or malignancy. The age of the patient, his history, past and present, the character of his surroundings, etc., in connection with the physical signs, carefully noted, will complete the diagnosis.

Treatment.—The typical ulcer, if not of excessive size, needs but little treatment; surgical cleanliness, with rest and protection of the parts, is sufficient.

Inflamed ulcer.—Thorough cleansing; absorbent gauze, wrung out of a one-per-cent. solution of carbolic acid, or three-per-cent. boric acid, covered with absorbent cotton and oiled silk or gutta-percha tissue, and a bandage to retain this dressing, with complete rest of the part, will probably meet all indications; or, if thought more agreeable to the patient, warm emollient poultices, followed by some astringent application, may be advised. I believe the treatment first outlined the better. If the patient be strong and plethoric, a saline purge, with avoidance of stimulants, may prove a benefit.

Sloughing ulcer.—Healthful surroundings, a generous diet, with tonics, like iron and quinine, warm, emollient, antiseptic dressings, and after the separation of the slough the dusting of the surface with iodoform every day or two, should be comprised in the treatment.

Weak or œdematous ulcer.—In this form of ulcer a stimulating treatment, both locally and generally, is most frequently required. The ordinary resin ointment, or, if that be inefficient, nitrate of silver, five grains to the ounce of water, should be brushed over the surface daily.

The *painful ulcer* requires soothing applications; it may be an application of cosmoline with the addition of carbolic acid, five grains to the ounce, or a linseed poultice with laudanum. Sometimes the destruction, by cauterization, of an especially painful spot, is necessary.

Indolent ulcers need stimulating applications, incisions through their edges, perhaps the application of a blister. They are rare in children.

When ulcers are *croupous* or *diphtheritic*, every source of infection should be guarded against. The membrane should be removed, and emollient, antiseptic dressings, as previously described, should be applied. Internally, with a generous diet, I have found benzoate of sodium of marked benefit.

Varicose ulcer.—Only rarely will we be able to remove the cause of this chronic ulcer. The excessive callus may in time get less; we may remove with a knife the tumor obstructing the veins; the deep cicatricial contractions may, by tedious operative procedures, be overcome. In all cases, however, the patient can be benefited by a judicious palliative treatment. The limb should be placed in a position which offers the greatest facility for the return of venous blood and lessens the arterial afflux. The œdema which complicates most of these ulcers will get less or disappear, the swollen, thickened skin will become normal, and active, healthy granulations will spring from the base of the ulcer. If absolute rest and immobility cannot be enforced, elevation of the limb at night, and elastic compression by the Martin bandage or the elastic stocking during the day, might be resorted to. I have seen great good come from a firm starched bandage. Some simple antiseptic dressing should be applied to prevent the accidental wound diseases, attacks of erysipelas, and the occurrence of septicism.

Strumous ulcers.—In this variety both constitutional treatment and local treatment are of vast import. The local treatment should be uncompromisingly radical. The undermined edges of the skin should be removed with the scissors, the granulations scraped off with the sharp spoon, and the base of the ulcer, if possible, dissected out. All the neighboring lymphatic glands which can be felt or seen should likewise be removed. The wounds left after the clean removal of the glands should be closed, after a thorough scrubbing with the bichloride-of-mercury solution, one to a thousand. Where primary union is impossible, iodoform becomes the sovereign remedy. It may be applied in the powdered form, and the wound-cavity filled with a loose absorbent tampon. I am in the habit of first injecting the tubercular foci with a solution composed of one drachm of iodoform to the ounce of sulphuric ether. After the evaporation of the ether, the iodoform remains in a thin, even film in every nook and corner of the treated part. These ulcers should be carefully watched until healing is complete; any return of the fungous growth calls for the same treatment. The unsightly scars seen upon the necks of patients who have suffered from this trouble may be dissected away, a linear scar resulting. Such patients should be placed under the best possible hygienic surroundings,—should have plenty of sunshine and fresh air, and nourishing food, such as milk, cream, eggs, and beef. If within the means of the patient, a course of sea-bathing, or a residence at the sea-shore, or removal to a dry, bracing climate, should be insisted upon. Of medicinal agents, tonics, such as bark and iron, iodide of iron and cod-liver oil, and, if digestion be poor, some bitter infusion, with pepsin or pancreatic emulsion, are to be administered. I have seen an almost specific effect from the long-continued use of small doses of the biniodide or the protiodide of mercury.

Syphilitic ulcers require for their treatment simple, nutritious food, frequent warm baths, mercury, iodide of potassium, and cod-liver oil; locally, a simple, emollient, antiseptic dressing.

In any ulcer following extensive loss of substance, nature may not be adequate to the task of covering the granulations with ample and healthy skin. In such cases skin-grafting, now a familiar procedure, should be resorted to. The method which I believe to give the best and most rapid result is Thiersch's. Remove all unhealthy granulations with the sharp spoon; arrest bleeding by pressing upon the wound gauze saturated with a six-per-cent. solution of chloride of sodium. Remove, with a razor, strips, one-quarter of an inch in width and of the required length, and in numbers to cover the ulcer fairly, of the superficial layer of healthy aseptic skin from the arm or thigh of the patient, and place them upon the ulcer side by side. Dress the part, and insure absolute rest for some days.

Amputation of a limb is occasionally necessary in extensive *circular ulcers* of the leg or at the ankle.

INFLAMMATIONS.

(CONTINUED.)

By L. DUNCAN BULKLEY, M D.

URTICARIA.

Synonymes.—Nettle-rash, Hives; German, Nesselsucht.

Definition.—An inflammatory, non-contagious affection of the skin, characterized by the more or less sudden development of wheals, associated with burning, tingling, and itching sensations.

History.—Urticaria may occur as a sudden outburst, almost furious in its character, involving much of the surface and causing great suffering; or it may appear more slowly, with the occasional development of a few wheals, which may come and go, even for a period of weeks or months. It very commonly appears in those supposed to be otherwise in perfect health, but may also accompany or follow other diseases, both of the skin and of other organs.

Etiology.—The etiology of urticaria is frequently very obscure: while in certain cases, especially of the more acute form, it will be caused by irritating food, such as fish, strawberries, pineapple, etc., or by an acute attack of indigestion, or by certain drugs, especially quinine, in a large proportion of cases it seems impossible to trace the eruption to any special cause, and the most rigorous attention to diet will fail to produce any beneficial effect upon the disease. It is recognized, however, that, in the main, urticaria depends upon disorders of the digestive system, and in children it is not infrequently caused by the presence of intestinal worms: in certain cases there will be a marked periodicity in the recurrence of the eruption, and it will be found that malaria is at the bottom of the trouble, which will then be checked entirely by quinine. In some cases the eruption may come from cerebral or spinal disease. External irritants are also often the cause of wheals, which may be produced by the bites of insects,—as mosquitoes, fleas, and bed-bugs,—also by puncture with the electric needle, and in those subject to the eruption the lesions can be produced at will by irritating the skin lightly, as with the finger-nail; in very susceptible skins it is possible even to draw figures on the skin with a blunt instrument which will shortly appear in elevated lines, often of considerable height and width.

Pathology and Pathological Anatomy.—The immediate causation of the wheal of urticaria lies, in all probability, in vaso-motor disturbance, which may have either a central, a peripheral, or a reflex origin. The essential element in the production of the wheals is a spasm of a localized tuft of blood-vessels, followed by relaxation and the consequent effusion of fluid, producing a localized œdema in the skin: the sensations of itching, burning, or tingling are the natural result of the compression of the sensitive nerves by the exudate, or may be in part due to the same direct or reflex irritation which excited the vascular spasm. The eruption disappears entirely after death, and microscopic sections taken during life have revealed little more than an œdema, with some transudation of leucocytes, and possibly some dilatation of the lymphatics.

Symptomatology.—The first symptom felt is commonly a general feeling of discomfort, with some burning or tingling of the surface, either in some particular spot or diffused over much of the skin. If there is the direct exciting cause of indigestion, there may be some febrile disturbance, but in the main no systemic symptoms are manifest, other than perhaps a little malaise, with a furred tongue and perhaps constipated bowels. The child scratches one and another portion of the body, and when these are examined there will generally be found the wheals already fully developed, or even fading away, and new ones may develop under fresh scratching even while being watched. But very commonly at the time of inspection the physician may not be able to detect a single lesion characteristic of the disease, but must rely upon the history and description as given by attendants, together with the scratch-marks which have been left after preceding eruptions.

The lesions of urticaria vary greatly in different individuals and at different times. The typical wheal consists of a firm, circumscribed, slightly-elevated spot or patch, from one-quarter to three-quarters of an inch in diameter, of an oval or rounded shape, and of a pinkish color at first, generally becoming white in the centre very shortly. In point of fact, however, they may be of any size and shape, large patches often being produced, and often assuming gyrate or fantastic shapes, largely due to external circumstances, as pressure of the clothes, etc. The separate lesions are commonly evanescent in character, if undisturbed, even disappearing in a few minutes, or they may remain in the same situation for some hours. When there is a single outburst of the eruption or one reproduced but a few times, it is spoken of as *acute urticaria;* when the disease-state is prolonged by continuous or successive crops of the lesions, it is spoken of as *chronic urticaria*, and the disease may thus last for months or years: other than the chronicity produced by the repeated outbreaks, there is no radical distinction to be noted.

Several varieties or forms of urticaria are described, and may be observed clinically.

1. *Urticaria Communis.*—This represents the eruption as ordinarily observed, where the wheals, of whatever shape or size, remain as such during their entire course, and subside and disappear, leaving only an erythematous blush for a while, which in turn fades, leaving no trace of the former lesion.

2. *Urticaria Papulosa.*—This variety, which was termed by older writers *lichen urticatus,* is seen much more commonly in children than in adults. In it there is, in addition to the wheal, which is generally about half an inch in diameter, a small solid papule developed in the centre, which remains after the subsidence of the wheal, and, consisting of organized lymph, may persist for a day or so. When brought for treatment, children will frequently present only the scratched papules, scattered here and there, principally on the extremities, around most of which the congested stain left by the faded wheal can be detected. This variety of the eruption generally assumes the chronic form, and, though the separate lesions may be of relatively brief duration, the child may be afflicted thereby for weeks or months.

3. *Urticaria Tuberosa.*—Occasionally the lesions of urticaria may take on great size, giving rise to the designation *giant urticaria,* some of them being raised up to the size of half a large walnut or a small egg; but this rarely if ever occurs in children.

4. *Urticaria Œdematosa.*—When the lesions are developed in situations where the tissue is lax, as about the face, there may be a very considerable amount of œdema, so that even the eyes may be closed and the tongue or lips may be greatly swollen: these are, however, generally very transitory, and do not call for active interference. There is reason to believe that the same process may take place deeper, in the fauces and trachea, or even on other mucous membranes.

5. *Urticaria Bullosa.*—In rare instances vesicles and blebs of greater or less size are formed in connection with urticarial wheals, by an augmentation of the congestive and exudative process producing the latter.

6. *Urticaria Hæmorrhagica.*—Occasionally there will be a hemorrhagic element manifested in cases of urticaria, and also in purpura urticarial wheals may sometimes be found among or around characteristic lesions of that disease, giving rise to the designation *purpura urticata* for this variety.

7. *Urticaria Factitia.*—This term is applied to cases in which, while there may be no very active symptoms of the disease, the skin is in such an irritable state that slight local irritation may give rise to wheals which correspond to the area excited. Thus, letters or designs can be traced with a dull instrument with a little pressure on the skin, and very shortly they will appear as white streaks with pink borders, which will remain standing out plainly for a longer or shorter time.

The minor varieties which have been referred to by authors under such terms as *urticaria conferta, evanida, febrilis, intermittens, miliaris, perstans,* *subcutanea, vesicularis,* etc., need not be dwelt on, their names expressing features which may be occasionally met with.

Diagnosis.—Very little difficulty will be encountered in diagnosing most cases of urticaria: the sudden appearance of the evanescent wheals, the peculiar burning and itching, and the irregular and more or less general distribution of the eruption are generally sufficient to make the diagnosis. The papular form of urticaria in children will frequently, however, resemble *seabies, papular eczema,* and *erythema multiforme;* but, if carefully examined, the remains of the wheals may usually be discovered surrounding the papule, and attendants will generally describe the larger lesions which appear suddenly, leaving the solid, scratched papule after they have faded. The eruption of urticaria, moreover, seldom occupies the places commonly affected in scabies, and of course none of the "furrows" pathognomonic of scabies can be found: it is well to remember, however, that the two are sometimes combined, and that the irritation of the burrowing insect in one place may excite reflexly an urticarial wheal in another. *Eczema,* especially in children, rarely remains papular very long, and its lesions are generally much more grouped and inclined to occupy the flexures than are those of urticaria. *Erythema multiforme* may sometimes be mistaken for urticaria, but its lesions are generally smaller, more abrupt, of a deeper red, slower in appearing, and far more permanent, the itching is much less marked than in urticaria, and there is little if any of the tingling and burning. They are also far more apt to be symmetrically developed, and to appear first or chiefly on the wrists or ankles.

Prognosis.—The prognosis of urticaria will differ greatly in different cases. Acute outbreaks caused by indigestion or irritating food commonly cease in a few days under appropriate treatment and a subsequent proper regulation of the life; but if neglected the acute may run into the chronic state, which may prove very rebellious. The papular urticaria of children will sometimes persist for weeks or even months in spite of the best treatment, but in the end the disease is curable, certainly in the very large proportion of cases.

Treatment.—Simple acute cases of urticaria may require little more than an evacuation of the stomach, if offending matter is still there, a moderate purge with castor oil or rhubarb and soda, and a little cream-of-tartar-water rather freely drank. But in chronic cases the utmost care in regard to diet, together with internal and external treatment, will often be required, combined with much patient study of the case. In some instances the most diligent attention to the diet, excluding one article after another, will fail to make much if any impression on the disease. This, however, should not lead to its being neglected, but during the entire course of the disease the diet should be plain, simple, and unstimulating, though abundantly nutritious, and with but a moderate proportion of sugar.

Alkalies internally are called for in a large share of cases, one of the best remedies being the well-known rhubarb-and-soda mixture, with peppermint-water after each meal, in doses suited to the age of the child,—sufficient to secure a moderately free action of the bowels daily; acetate of potassium may be added to this with good effect in many cases. Alternating with this, iron and arsenic, or cod-liver oil, will generally be found sufficient for the cure; the hypophosphites are also frequently called for, and quinine, even in children, may sometimes be given in free doses with the best of effect.

Locally the free use of the following lotion will generally be found to give relief, it being sopped abundantly over the affected surface and allowed to dry upon the skin: ℞ Pulv. calaminæ præp., ʒi; zinci oxidi, ʒii; acidi carbolici, ʒss; glycerini, ʒiii; aquæ calcis, ʒiv; aquæ rosæ ad ℥iv. M. The application may be made several times daily, or, when desired for the relief of the itching, day or night: if the surface becomes dried, a light inunction of carbolized cosmoline (gr. v–x ad ℥i) will give relief. Menthol in solution thus (℞ Menthol., ʒss–ʒii; spts. vini rectif., ℥i; glycerini, ℥i; aquæ, ℥iv. M.) is also serviceable. In some instances a powder gives the most relief, and the following, well rubbed on the skin with the palm, forms a very agreeable application: ℞ Chloral. hydrat., gummi camphoræ, āā ʒi. Rub together till liquid, and add pulvis amyli, ℥i. M. In more chronic cases alkaline baths are of service, made as follows: ℞ Potassii carbonat., ℥viii; sodii carbonat., ℥vi; sodii biborat., ℥iv. M. Sig. Use from two to four teaspoonfuls to each gallon of water, with an equal quantity of starch. After the bath, which should be pleasantly warm, the surface of the body should, when dry, be thoroughly anointed with the carbolated cosmoline: the lotion may be used in conjunction with this when required to relieve irritation.

URTICARIA PIGMENTOSA.

Mention may be made here of a rare affection under the above name, which thus far has been always observed to begin in children, frequently as early as during the first few months of life, seldom later than the third year. The eruption begins as an urticaria, the lesions appearing in the course of a night, but, instead of disappearing as in ordinary urticaria, they remain as solid, buff-colored tubercles, or infiltrations, somewhat resembling xanthoma, even for years. New crops may come out from time to time, each lesion seeming acute, but most of them result as described, and thus the surface becomes more and more covered. Two classes of cases are described,—the pruritic, which is the more common, and the non-pruritic.[1] The lesions may affect any or all portions of the body, but are most abundant on the neck and trunk, next upon the limbs, face, and head, and occasionally on the palms and soles. The real nature of the complaint has never been satisfactorily determined, and no treatment has ever proved of much avail in it.

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[1] Radcliffe Crocker, Diseases of the Skin, London, 1888, p. 87.

ERYTHEMA MULTIFORME.

Definition.—An acute inflammatory disease, characterized by the more or less symmetrical development of variously sized and shaped lesions, principally erythematous in character, but also producing papules or flattened tubercular elevations, and occasionally vesicles and bullæ.

History.—The eruption is very commonly preceded by a certain amount of constitutional disturbance, generally very slight, but sometimes so severe as to suggest a graver malady. There may be some stomach-disturbance, with malaise, some headache and backache, and usually more or less pains in the limbs, especially in the joints: the temperature may run from 100° to 104° F. and the pulse be considerably quickened. In many cases, however, the general symptoms are so slight that the eruption is the first and possibly the only sign of disease which attracts attention.

Etiology.—Little is definitely known in regard to the true etiology of erythema multiforme, although it is pretty well established that both this and erythema nodosum stand in very close relation to the rheumatic diathesis, if indeed they are not expressions of the action of this poison on the skin, even when there are no other marked rheumatic symptoms present. It is more common during the spring and autumn, and attacks may be induced by chilling the surface when overheated. The eruption is more frequent in females than in males, and may attack any age, and, while more commonly seen in young adults, is not a rare complaint among children: teething may act as an exciting cause. Among other agencies capable of producing the skin-lesions classed under multiform erythema must be mentioned the diphtheritic poison, also those of variola, cholera, typhoid and typhus fevers, and likewise certain drugs, notably copaiba, quinine, the preparations of iodine, etc.; although these eruptions do not really belong here, but under their proper etiological head.

Pathology and Pathological Anatomy.—From whatever cause the eruption may arise, it appears that the lesions are caused by vaso-motor disturbance, resulting in capillary hyperæmia with subsequent stasis and exudation, with the escape of leucocytes and blood-coloring matter, as is shown by the staining following the disappearance of the eruption: in some instances actual hemorrhage from the blood-vessels takes place. The amount of exudation varies greatly, being very slight in the erythematous and slightly papular forms, and at other times so great as to cause considerable elevation in the lesions, or even to raise the epidermis in vesicles or bullæ. Examined microscopically, the tissues exhibit the signs of inflammation of the upper part of the corium.

Symptomatology.—The appearances of the eruptions which are classed under erythema multiforme by modern observers are so different at times that it is difficult to describe briefly their characters: they may vary from a true erythematous blush to a solid lesion of some little size, and even to the production of vesicles or blebs in rather rare instances. These various lesions may appear blended at the same time, or in successive crops or groups. The character of the eruption is that of an inflammatory exudative disorder, whose type is a superficial or erythematous lesion, in the main short-lived, which, however, rather tends to some localized infiltration of tissue. The extent of the eruption also varies greatly in different cases, from a comparatively few spots up to a quite generalized eruption, causing very considerable distress. Erythema multiforme seldom fails to appear on the backs of the hands, and in a considerable proportion of cases is confined to this region and the forearms, the next most frequent locality being the ankles or the tops of the feet. Several quite well defined classes or groups of cases can be made out, although frequently the eruption passes from one form to another.

1. *Erythema Papulatum.*—This may be said to be the typical form of the eruption, where groups of deep-red papules from the size of a pin-head to that of a small split pea appear quite suddenly, generally on the backs of the hands or wrists. These at first are almost wholly erythematous, disappearing under pressure, though leaving a slight stain, and may remain such throughout their entire course; they may continue to increase in size and gain in solidity and height, although never much raised above the surface: when they attain the size of a split pea, the name *erythema tuberculatum* has been given to them. There is some little burning pain in the affected locality, and they may be tender to the touch. The eruption may remain in this stage for some days, and then fade, new spots appearing, or the process may go on to the production of the next variety,—

2. *Erythema Circinatum.*—The separate papules increase rapidly in size, the centre begins to pale or become of a purplish hue, and a circle or ring may be formed even of an inch or more in diameter,—*erythema annulare* or *erythema centrifugum*,—and sometimes, from the fading and changing hues caused by the different stages of the process, zones of color, from purple to pink, may be formed,—*erythema iris*. When several of these rings touch one another, variously-shaped figures may be formed,—*erythema gyratum*, *erythema figuratum;* and when the margin of the eruption is sharply defined and raised, advancing, it may be, over a large surface, even within a few days, the name *erythema marginatum* has been given to it.

3. *Erythema Vesiculosum.*—When the process is more acute and sudden, the effused fluid cannot find place within the integument, but rises to the surface, and, being arrested by the firm horny layer, raises it into vesicles of greater or less size, which may be irregularly located or may be grouped in clusters; sometimes these are arranged in a circle on the advancing edge of an erythematous spot, and it may even happen that another, inner circle

of vesicles is developed, often with a single one in the centre,—*herpes iris.* In some instances the collection of fluid may be so great that even bullæ are formed,—*erythema bullosum,* which may then strongly suggest pemphigus.

Diagnosis.—Some of the forms of erythema multiforme may at times give considerable difficulty in diagnosis, and the determination must often be made by exclusion. But the multiform and varying character of the eruption, its symmetry and localization, the comparatively slight subjective symptoms of itching and burning, and the more or less pronounced rheumatic symptoms often present, are generally sufficient to distinguish it.

The papular form may be mistaken for *urticaria, lichen planus, papular eczema,* and possibly for a *syphilide.* Urticarial lesions are more sudden in their appearance, attain their full size at once, and have much more itching and burning; the papules of *lichen planus* are more distinctly demarcated, flat and shiny on top, with a depressed centre, and more purplish pink than those of erythema papulatum; *papular eczema* is far more itchy, with more acuminate papules; while the *syphilide* would present other and general symptoms sufficient for the diagnosis.

The circinate form of erythema resembles somewhat a *tinea circinata,* but the latter is much more slow in spreading, and is always more or less scaly from the first, and the parasite can always be found microscopically in the scales.

The vesicular and bullous forms are distinguished from *pemphigus* by the amount of erythematous and inflammatory elements and the multiformity of the lesions, the bullæ of the latter affection commonly rising from a more healthy surface.

Prognosis.—This is invariably good, the eruption generally running from one to three or four weeks, and disappearing without leaving any trace, other than a staining which remains but a short time.

Treatment.—Comparatively little treatment is required, other than such as may be called for to meet general symptoms: a slight saline laxative, as cream-of-tartar water, or a mild alkali, as acetate of potassium, aids somewhat in dissipating the disease. Locally, a calamine-and-zinc lotion such as that mentioned under urticaria, or a dusting-powder of powdered starch with a little camphor and oxide of zinc, gives all the relief required.

ERYTHEMA NODOSUM.

Most writers agree now that this eruption is in reality but a magnified and somewhat different form of erythema multiforme, for, while in its most pronounced manifestations it is striking and different from the other varieties already mentioned, some of its lighter lesions cannot be distinguished therefrom.

In its characteristic form erythema nodosum presents oval or roundish, node-like swellings, pink in color and tender to the touch, along the tibiæ or ulnæ and elsewhere. These may vary in size from that of a chestnut to that of half an egg and merge insensibly into the surrounding tissues; they feel at first hard and tense, but later seem to soften, and give almost the sensation of fluctuation, but they never suppurate unless injured. The tumors last from one to two weeks, and gradually fade away, leaving a dusky, bruise-like stain, whence the name *dermatitis contusiformis.*

There is generally more or less general disturbance in these cases, headache and a coated tongue, malaise, and some aching pains in the limbs, and some children may present a decided elevation of temperature.

The etiology, pathology, prognosis, and treatment are practically the same as in erythema multiforme.

HERPES ZOSTER.

Synonymes.—Zona, Shingles; German, Gürtelkrankheit.

Definition.—An acute inflammatory eruption, exhibiting groups of vesicles upon an inflamed and very sensitive surface corresponding to a definite nerve-tract, and accompanied by more or less neuralgic suffering.

History.—Zoster usually comes on with a neuralgic pain, which may be very acute, in the part about to be affected, and sometimes there may be a little febrile disturbance, but generally without any other sign of ill health: not uncommonly a mustard plaster or other counter-irritant is applied, and the eruption which appears is wrongly charged to this cause.

Etiology.—No very definite statements can be made in regard to the true etiology of this eruption: the pathological condition which appears to be at the base of it is well known, but why nerve-inflammation should at one time produce the eruption and at another fail to do so cannot be told. From the fact that it usually occurs but once during a lifetime, and from the occasional appearance of several cases together or in succession, some have been led to regard it as an infectious disease, while others have attributed it to atmospheric influence; but no data have been recorded which give sufficient support to either supposition. Hutchinson, having observed a number of cases develop while patients were taking arsenic, suggested that this could cause the eruption, and others have repeated the observation. Atmospheric changes, cold draughts, and exposure to wet can cause the nerve-inflammation associated with the eruption. Zoster is quite common among children, and generally runs a mild course in them.

Pathology and Pathological Anatomy.—The skin-lesions in zoster are the direct result of irritation of the nerve or nerves distributed to the affected skin. This irritation may exist in any part of the course of the nerve, but is most commonly found in the spinal ganglia, and a number of autopsies have demonstrated interstitial neuritis of the posterior or sensory ganglion, as was first shown by Bärensprung. But later researches have also demonstrated this to be healthy in certain cases, while neuritis existed in other portions of the nerve: cases also are reported where there was hemorrhage into the Gasserian ganglion, also into the cauda equina, in a case of crural herpes, also where there was disease or injury of the spinal cord, and many other conditions inducing nerve-irritation and inflammation. The nerves near the eruption have been found to be the seat of a perineuritis. The local disease in the skin consists in an inflammatory process beginning in the rete, although the papillary layer shows also many signs of inflammation. The exuded fluid forces its way among the cells of the rete, stretching them into fibres which at first intersect the vesicles: later these all melt away, and the vesicle becomes unilocular, containing rete-cells, pus-corpuscles, and serum. The epithelial covering of the vesicles is firm and adherent, and has but little tendency to rupture, except under harsh usage: when it dries down without rupture scarring seldom occurs, but if this is broken the surface is apt to ulcerate superficially and permanent cicatrices are left.

Symptomatology.—The eruption of zoster is developed along the line of some distinct nerve-tract or area, most commonly about the trunk, and, with the rarest exceptions, is always confined to one side of the body, the explanation being found in the pathology of the disease. The eruption, however, often laps a little over the middle line, owing to the interlacing of the nerve-filaments of the two sides of the body, but cannot continue around the body: cases have occurred, however, where there has been a double zoster at the same line, thus making a complete circle or girdle around the body, and these cases are not any more dangerous than others, notwithstanding the popular superstition to the contrary. The eruption may also follow any nerve-line, and is not uncommonly seen along the limbs, and, especially in adults, along the tract of the cranial nerves.

The separate lesions begin with one or more inflamed patches, tender to the touch, as though burned or scraped, and giving the sensation of heat and burning to the patient. Within a few hours minute points can be seen, which soon develop into vesicles, which take a flattened shape, and may be closely set or scattered; the eruption is developed first near the root of the nerve, the patches or the more distal portions following, even some days later; in some places the eruption may stop short at the erythematous stage. The disease takes from three to ten days to reach its height, sometimes a little longer, and about the same length of time for the lesions to dry up, although often the crusts may remain adherent for three weeks or more, and, if the surfaces are irritated, ulceration may follow, which will take a longer time to heal. The amount of eruption varies greatly in different cases: in some there will be a broad band over the affected surfaces, with the groups of vesicles almost or quite touching one another; in other cases the inflamed patches and groups of vesicles may be small, and separated some distance apart, and occasionally but a single group or two will appear; perhaps with some erythematous redness between.

The eruption has received various names according to the locality attacked. On the trunk it receives such names as *zoster pectoralis* and *abdominalis;* on the head, *zoster faciei, frontalis, nuchæ,* and, when affecting the first branch of the fifth nerve, *zoster ophthalmicus:* for the extremities we have *zoster brachialis, lumbo-femoralis, cruro-genitalis,* etc. In every instance the disease is one and the same, presenting somewhat different appearances according to the locality. It very rarely affects the hands and feet,—it may be said never, except when there is an abundant eruption on parts nearer the trunk.

Diagnosis.—This should seldom be difficult when the features of the disease are all well borne in mind: the one-sided character of the eruption is always a striking feature, as also the grouping of the lesions along nerve-tracts; even early in the course of the disease, the erythematous patches, tender to the touch, should suggest the diagnosis, while when the flattened grouped vesicles are formed the appearance differs materially from that presented by any other skin-affection.

Prognosis.—Zoster is really a self-limited disease, and, even under adverse circumstances, offers a favorable prognosis. But patients should be made aware that scarring may result, which in very rare cases about the face may prove troublesome, and, when the eruption is located about the eye, that organ may be endangered; a certain amount of neuralgia may also persist after the eruption is cured. Fortunately, neither of these features is common in children.

Treatment.—Little or no internal treatment is required, other than to meet symptoms, as internal medication can influence but slightly if at all the course of the disease: the neuralgia, both before, during, and after the attack, may require nerve-tonics, especially quinine. Locally the simpler the dressing the better. Plasters and ointments are to be avoided in the main, as they tend to break the vesicles and to cause an open sore. The aim of treatment should be to have the eruption dry down, and the vesicles form a scab, which should remain attached until it falls spontaneously. If left unprotected, the clothing and external forces are very apt to rub and chafe the part, and not only cause much suffering but also break and tear the lesions and so delay the recovery.

In most cases the simplest and best treatment is to powder the affected surface well with very fine starch or rice-powder, with which a little morphine and zinc oxide are mixed if desired, and then to dust thickly also a piece of fine linen or muslin and bind it very firmly over the surface, in such a manner that it shall not move, and that the clothing may slip over it; in this way all direct friction of the part is avoided. Sometimes a thin layer of cotton batting may be well dusted with the powder and placed beneath the bandage, which is best sewed on, in order to have it drawn as tightly as possible. When this dressing is comfortable and remains in position, it may be left intact even for a number of days, and when taken off the eruption will be found to be quite dried up. If the vesicles should break and the cloth stick to them, it will have to be gently removed by soaking, and then fresh powder applied, and a very thin layer of absorbent cotton, well powdered, laid on top, which may be allowed to dry on and may even be left until the surface is entirely healed.

Writers recommend many local applications in zoster, but the plan here described, if faithfully and intelligently carried out, leaves little to be desired. Sometimes, if the pain is excessive, anodyne liniments or ointments may be required, and the calamine-and-zinc lotion previously referred to will frequently be found of service in allaying the burning heat. Where the pain is great, the galvanic current applied directly over the lesions will give relief, and will occasionally even seem to arrest in a measure the eruption itself.

PEMPHIGUS.

Definition.—An acute or chronic inflammatory disease of the skin, characterized by the successive formation of bullæ of various sizes, generally upon a slightly-inflamed base.

History.—Much discussion has arisen from time to time in regard to pemphigus and the eruptions which should be thus classed, and advancing knowledge in dermatology has separated many conditions formerly classed under this head; such are the bullæ already described under erythema multiforme, also those caused occasionally by drugs, those found in syphilis, leprosy, etc., so that this designation is now pretty distinctly limited to a relatively small class of cases; but the disease still exists as an entity, and may be met with entirely disassociated from other skin-affections. Three quite distinct forms may be recognized, which will be described later,—namely, *acute pemphigus*, *chronic pemphigus*, and *pemphigus foliaceus.*

This disease is usually ushered in, especially in children, with more or less constitutional disturbance, thirst, loss of appetite, and some fever; during the course of the disease there may be diarrhœa, and some constitutional disturbance often recurs with each first attack of the eruption: in fatal cases the strength fails gradually with the continued production of the bullæ, and death may occur from exhaustion early in the disease, especially in infants, or may follow a prolonged existence of the eruption.

The lesions of pemphigus usually appear without much if any premonitory sign on the affected part, and with comparatively little sensation, but when developed give rise to a tense, sore feeling, and when the bullæ are ruptured, which occurs early, the surface is apt to be very painful. They may appear singly or in crops and scattered indifferently over every portion of the body and extremities. In size they may vary from that of a small pea to that of half a large egg, generally rising in globular form abruptly from a slightly-inflamed base.

Etiology.—Pemphigus is essentially a disease of lowered vitality, and most probably of nervous origin: in adults it not uncommonly follows nerve-exhaustion, and is seen in connection with certain diseases of the brain and spinal cord. It is much more common in infants and children than in adults, and is said to be equally frequent in both sexes. No definite cause can be found in diet, in atmospheric or other conditions, nor in any dyscrasia; syphilis may produce a bullous eruption, especially in children, but this is not classed as true pemphigus. The disease is a rare one.

Pathology and Pathological Anatomy.—Little is known as to the real pathology of pemphigus, but from autopsies made by Schwimmer[1] and Babes, with microscopical examination of the spinal cord in two cases, it would seem probable that the origin of the disease may be located in the cord: they found sclerosis of the posterior columns in both cases, with atrophy of the anterior horns, there being medullary changes throughout almost the entire length of the cord.

The skin-lesions have been examined microscopically by a number of observers: the bullæ are found more superficially seated than in herpes or eczema, the covering being formed of the horny layer with some of the rete-cells, and the base resting on the deeper cells of the rete and the corium; the stretched cells of the rete and ducts of glands form strings or fibres here and there, which may depend from the roof or may connect it with the base, especially at the sides of the bulla. Beneath the lesion the usual signs of inflammation are seen in the corium, and also a parenchymatous neuritis in some cases, similar to that observed in other inflammatory lesions, so that no causative importance is attached to it.

The contents of the bullæ are usually alkaline, strongly albuminous, and contain phosphates: many other ingredients have been described as found in different instances, but no pathological significance has been given to them.

Symptomatology.—The three forms of pemphigus present such different phenomena that they require separate descriptions.

Acute Pemphigus.—This is the form of disease which is seen principally in children, and as *pemphigus neonatorum* often proves very fatal, occasionally appearing almost as an epidemic in lying-in institutions, occurring principally in feeble and ill-nourished children and amid unsanitary surroundings. Cases of acute pemphigus may differ very greatly in severity, from mild cases where a comparatively few bullæ develop on different parts of the body, the disease running a favorable course in two or three weeks, to severe and fatal cases, which may take on a gangrenous aspect,—*pemphigus gangrænosus,*—the child perishing in ten or twelve days. Pemphigus is apt to follow convalescence from acute febrile diseases, as scarlatina and measles, and in young infants it has been ascribed to the practice of putting them into too hot a bath.

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[1] Schwimmer, Die neuropat. Dermatosen, Wien, 1883, p. 149.

Chronic Pemphigus.—This is the more common form in adults, and occurs more or less frequently in children. The eruption generally begins quite acutely, with the outburst of one or several bullæ, which may appear suddenly as small, clear, globular vesicles, almost as if produced artificially with a drop of scalding water; they enlarge rapidly, and in a single day may attain the size of half an egg. The lesions seldom touch one another, and never run together.

In some cases the crops of vesicles will appear in very rapid succession, each day producing a number; in other cases their development will be more tardy, and one crop will almost dry off when a fresh one will appear, and so the disease may be prolonged indefinitely. Lesions may appear upon the lips and tongue and in the buccal cavity and pharynx, and render deglutition and talking very difficult: from attacks of coughing and diarrhœa which may occur in these cases, it seems probable that the same lesions may be formed also on the deeper mucous membranes. The amount of distress which may be occasioned by this disease is very great, the sufferer being often unable to lie in any position or to make any movement without tearing the raw surfaces left after the bullæ. If not checked by treatment, these patients succumb, perhaps after months, worn out by constant distress and by a diarrhœa which cannot be checked.

Pemphigus Foliaceus.—Seen at its height, this disease would hardly be recognized in its true nature, as it then presents only a reddened surface upon which are epidermal laminæ attached at the edge, with no trace of bullæ, the epidermis not being able to hold together to form such. But early in the disease bullæ are observed, which are more flaccid than in the preceding form, and which readily burst. Beginning with a few blebs, the eruption may extend until the entire surface of the body is covered with the scaly condition just described. Happily, this is a very rare affection, and seldom occurs in children.

Diagnosis.—This may sometimes present no little difficulty, and care should always be exercised to eliminate the other conditions in which bullæ may appear. Thus, we may have them from *artificial causes*, as burns, chafing, or irritating external applications, also from certain *drugs taken internally;* they are also seen sometimes in *erythema* and *urticaria*, also in *eczema*, and occasionally about the hands and feet in *scabies; herpes zoster* and *herpes iris* may present quite large bullæ, as also *varicella* and *impetigo contagiosa;* they are likewise sometimes seen in *erysipelas*, and, finally, are not uncommon in *infantile syphilis.* Due care, however, in recognizing the features of the disease and in excluding those mentioned should establish the diagnosis with certainty in most cases without much difficulty.

Prognosis.—This will vary greatly according to the individual case, and must always be given very guardedly, for few diseases run a more uncertain course than pemphigus. Relapses may come when least expected, and no reliable indications can be stated from which one might judge certainly of a favorable course or issue of the disease. It is by no means, however, fatal even in a large proportion of cases: the large majority recover, both of children and of adults.

Treatment.—But one remedy appears to have any controlling influence over pemphigus, and in many cases this proves a most valuable resource, and that is arsenic. But to be of real value it should be given freely, frequently, and fearlessly. It is especially serviceable in children, and is remarkably well borne by them. It should be given, diluted in at least one-quarter or one-third of a goblet of water, every two or three hours, in doses increasing in quantity until the disease yields or until some signs are given that it disagrees with the patient. Usually diarrhœa will be the first sign of disagreement; and even then, if the disease is not checked, it may often be continued freely, and this action may be checked by adding a little opium, which also acts favorably on the disease. The results following this plan in many cases are astonishing. Attention should be given to the general state of the patient and supporting treatment given, but alcohol is prejudicial to the eruption.

Locally, great difficulty is often experienced even in making the patient tolerably comfortable. The blebs do better if punctured near their base with a fine needle in one or two places and the serum allowed to ooze out, and the covering made to rest on the base of the bulla: this should be preserved in every case as long as possible. Sometimes thin layers of absorbent cotton are the very best dressing; in some cases a damp application, as the calamine-and-zinc lotion, gives great relief; at other times a dusting-powder covered with absorbent cotton serves the best. When there is a raw surface to which the cotton is not grateful, a very mild ointment of oxide of zinc, half a drachm to the ounce of rose-ointment, or cucumber-ointment, with half a drachm of tincture of camphor or a few drops of carbolic acid to the ounce, will afford most relief. Care must be exercised in putting morphine in these ointments, as the raw surfaces readily absorb it, and serious results might follow from its application over too large an area. When there is much denuded surface, comfort has been obtained by a continuous warm bath, in which the patient may lie on a mattress, Hebra keeping some patients in this condition for many months, in comparative ease; but much use of water, except in this manner, is prejudicial in these cases, and rather tends to the development of new blisters.

PRURIGO.

Synonymes.—True prurigo (of Hebra); German, Juckblättern; French, Strophulus prurigineux.

Definition.—A chronic inflammatory affection, exhibiting shotty papules, pale red or of almost the color of the skin, with intense pruritus, and subsequent thickening and pigmentation of the integument.

History.—Prurigo is exceedingly rare in this country, although very common in Austria; but very many cases are wrongly so considered which in reality are quite different affections. The distinction should be clearly drawn between prurigo and pruritus; the former is a distinct disease, while pruritus or itching is a symptom or element in many diseases, and also occurs as an independent condition. Nor has prurigo anything to do with the eruptions caused by pediculi; all writers agree now in confining the name to a distinct affection, first clearly defined and isolated by Hebra, from whom most of our knowledge concerning it is derived.

Prurigo usually begins in early childhood, even within the first or second year, according to Kaposi, and then in the form of an urticaria.

Etiology.—Nothing definite is known in regard to the causation of prurigo. It is not due to any external causes, as parasites, nor is it from any special dyscrasia, except that it is most often seen in debilitated, scrofulous subjects. It is more common in males than in females, and generally begins very early in life, from the second to the seventh year; according to Hebra, it never develops primarily in adult life, but in every case it has persisted, though in mild form, from childhood; it is said to be worse in winter, and to undergo exacerbations with the change of seasons.

Pathology and Pathological Anatomy.—Little is known in regard to the real pathology of this disease. When examined microscopically the papules present much the same appearances as those found in the lesions of eczema: the disease is mainly seated in the papillary layer and in the deeper cells of the rete, and nothing has been found to explain the intense itching almost constantly present. Hebra believes that the formation of the papule is primary, and that the presence of the exuded plasma gives rise to the pruritic sensations; others believe that the eruption is a trophoneurosis, but no changes in the nerves have yet been demonstrated.

Symptomatology.—Two forms, varieties, or, more properly, degrees of this disease are recognized,—*prurigo ferox*, and *prurigo mitis*.

Prurigo ferox is the form seen in Vienna, and rarely if ever encountered here. Beginning with a mild eruption, it gradually increases in severity until the suffering may be atrocious. When fully developed, the skin of the affected part—more commonly the extensor surfaces of the lower limbs, occasionally also those of the upper extremities—is found to be thickened, with many solid papules, and more or less covered with the results of scratching, crusts, and scales; the torn papules exude a drop of serum, and momentary relief is afforded thereby; but the itching returns, and by repeated scratching the skin may be greatly torn; in severe and old cases the inguinal glands are enlarged. The flexor surfaces are spared, and the axillæ, bends of the elbows, groins, and popliteal spaces will be smooth and white, when the rest of the surface is torn and pigmented.

Prurigo mitis is not necessarily an early stage of the preceding, but may remain from first to last in a form warranting this name. Beginning in early infancy or childhood, with more or less urticarial features, the solid papules, almost flesh-color, continue to form here and there, mainly on the extensor surfaces, with great itching. The condition may be much improved by the advent of warm weather, when the skin perspires, which it does not otherwise do, but with return of colder weather the itching increases and new lesions form, with greater severity of itching, and so the disease may vary from time to time, never quite yielding, but becoming more and more inveterate. These cases are rare, and many which might be supposed to be such will be found to be only chronic papular eczema.

Diagnosis.—The essential features of the eruption are, the hard, isolated, non-inflammatory papules, seated on a harsh, dry skin, mainly on the extensor surfaces of the extremities, with intense itching following rather than preceding the appearance of the papules, which may often be felt before they become visible, the early beginning and obstinate continuing of the eruption, and its varying with the changing seasons. The affections to be chiefly differentiated from it are *pruritus*, *eczema*, *papular urticaria*, *scabies*, and *pediculosis*.

Prognosis.—This is always doubtful: Hebra declared that well-established cases of prurigo were incurable.

Treatment.—This is largely to be conducted on general principles, as no single remedy or particular line of treatment has ever yielded great results. Every element conducing to the improvement of the general health should be carefully looked to, and the utmost endeavor made by means of improved diet, hygiene, etc., to raise the general tone: these are to be aided by such remedies as cod-liver oil, iron, and phosphates, with careful attention to the excretions from the kidneys and bowels. Locally the measures serviceable in eczema will be found of most value, especially alkaline baths, tar, and such preparations as soften the surface and remove the outer layers of the skin.

PSORIASIS.

Synonymes.—Lepra Willani, Lepra vulgaris, Alphos, Dry or Scaly tetter; German, Schuppenflechte.

Definition.—A chronic inflammatory affection of the skin, exhibiting dry, red, slightly-elevated patches or spots, of varying size and shape, generally circular, covered with a greater or less quantity of dry, white, silvery scales, heaped together, and occurring chiefly on the extensor surfaces.

History.—Psoriasis represents a definite, clearly-defined disease of the skin, and the term should not be applied to scaly stages of eczema, nor to the large papular eruption of syphilis which sometimes resembles it. Some of the older writers, as Willan, applied the term *lepra* to a certain form of this disease, and confusion has sometimes arisen thereby, as this latter name is now employed to designate true leprosy, or *elephantiasis Græcorum*, with which the disease under consideration has nothing in common.

Psoriasis is one of the more common diseases of the skin, but it is not so frequent as is generally supposed : thus, while eczema forms thirty-three per cent. of all skin cases in most statistics, psoriasis forms hardly four and a half per cent. The eruption may begin at any time of life, but it rarely commences after the fiftieth year : more commonly it makes its first appearance during early adult life or childhood, and it has been observed as early as eight months. Very great variations are seen in the amount and degree of the eruption, from a few, small, and apparently insignificant scaly papules seated mainly on the extensor surfaces of the extremities, to a diffused eruption of large patches covering much of the surface. In children, however, the eruption is rarely very extensive, although there may be a large number of small lesions pretty well distributed over the body and limbs, and not infrequently upon the face.

Etiology.—In most cases no sufficient cause for the eruption can be found. It is not produced by contagion, for, although it has been claimed that a microscopic vegetable organism has been found, the clinical facts are strongly against such an hypothesis ; nor has the eruption ever been induced artificially at will. External irritants cannot produce it, although in children it will sometimes be found to develop directly after vaccination in such a manner as to suggest such a causation : it is also not infrequently seen to follow closely after attacks of scarlet fever, measles, and chicken-pox, but it may also develop after any depressing cause, as a prolonged sickness, and in women it has been seen to follow on parturition and lactation. In large statistics it will be found to attack males and females in almost exactly the same proportion. The seasons of the year have almost no definite effect in its production, although a greater number of cases are seen to appear first in the spring and in the fall, at which times the eruption is generally worse in all cases : it is usually better in summer.

Although psoriasis appears to be a disease of internal origin, very little is known in regard to its etiological elements in this direction. It appears alike in the rich and the poor, in all stations and occupations of life, and in those living on widely different diet in various countries : no single element or class of diet seems to be capable of its production, nor can it be surely cured by any particular course of living. Those exhibiting the scrofulous diathesis are less subject to it than those of more full habit : it seems particularly to flourish in those of ruddy complexion and presenting more or less of the gouty diathesis ; and, as will be seen when considering its treatment, it is in recognizing and treating properly this condition that most gain can often be had in handling the disease.

Aside from the gouty element just alluded to, the only other well-established fact in regard to the etiology of psoriasis is that of *hereditary transmission*. But this is by no means so important a factor as is often supposed : while it is not infrequent to find cases where the disease had existed in parents, in the greater number of instances, even in the more intelligent patients seen in private practice, no such antecedent history can be discovered, while very many psoriatic patients have perfectly healthy children; very rarely will all the children of a family be affected, and it will constantly be found in a single member, though instances are on record where almost all the children and one of the parents have the disease. It will not infrequently be found to skip one or more generations, and occasionally to alternate with gout, asthma, etc.

Pathology and Pathological Anatomy.—The pathological anatomy of this disease has been very thoroughly studied by a number of observers, but throws little or no light on the true nature of the affection. The following is briefly the condition found: a hyperplasia of the Malpighian layer, whereby the interpapillary masses are greatly increased in size, both in length and in breadth; a corresponding increase in the length and size of the papillæ, so that in certain specimens the two interdigitate in a striking manner; an enlargement, both in length and in breadth, of the blood-vessels, especially those in the upper part of the corium and papillæ, and a moderate cell-infiltration around them; changes are also seen in the hair-follicles, consisting primarily of hyperplasia of the root-sheath, presenting finger-like outgrowths similar to those described in the rete; according to Crocker,[1] "the cell-effusion extends downwards around the sweat-ducts, and the glands also exhibit cell-proliferation, blocking up the lumen of the lobules and producing the appearance of the whole gland being a uniform mass of cells;" in addition to these there is a great increase in the horny layers of the epidermis, which, according to Crocker, are separated by "enormous numbers of minute, circular bodies, with a central dark spot, which lie in loose clusters between the separated layers, but which also exist in dense masses lying horizontally in the still adherent horny layers below," suggesting micro-organisms; but nothing is yet known as to whether these have any bearing on the disease.

The main difference of opinion in regard to the pathology of the disease is in reference to the changes in the rete Malpighii, which are seen alike (though with varying intensity) in minute new points and in old patches: the question is, as to which of them are primary and which are secondary. Older observers considered psoriasis an inflammatory affection, and regarded the hyperplastic changes in the rete as secondary to this. Robinson[2] and others believe this latter to be the primary disorder, the inflammatory changes in the corium following and being dependent thereupon: this agrees in a measure with the views enunciated by Tilbury Fox,[3] several years ago, that "the disease consists primarily and essentially in a misbehavior of the cell-elements themselves, a perversion of the ordinary cell-element of the epidermis." The matter is by no means settled, nor has any light been thrown on the question as to what is the ultimate factor in the production of the changes described; whether the bodies observed by Crocker are microbes which have an etiological bearing, or whether the trophic nerves play a part in the epithelial hypertrophy, or whether nerve-influence first excites the capillary alterations, cannot be now more than conjectured.

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[1] Crocker, Diseases of the Skin, London, 1888, p. 182.

[2] Robinson, A Manual of Dermatology, New York, 1884, p. 391.

[3] Tilbury Fox, Skin Diseases, London, 1873, p. 264.

Symptomatology.—However varied the appearances are which fully-developed cases of psoriasis may present, the individual spots always appear first, singly and separate, as small points, not larger than a pin's head, of a pale-red color, slightly elevated, and resting upon otherwise apparently healthy skin; these points rarely come singly, but a number develop at the same time, though not in groups, except that particular localities are most likely to be affected at once. The places most frequently attacked are the extensor surfaces, the flexor surfaces being generally spared, or at least seldom if ever affected before the eruption has appeared very abundantly elsewhere. The palms and the soles are very rarely invaded,—indeed, so seldom that some have denied the occurrence of true psoriasis in this locality, where the syphilitic eruption, which so closely simulates it, is very common. The eruption may occur alone, or first, on the scalp, but rarely appears on the trunk until the extremities are affected. The eruption of psoriasis is generally developed with a remarkable symmetry on both sides of the body.

The minute papules of psoriasis enlarge peripherally, sometimes very quickly, but generally at only a moderate pace, until they attain various sizes, and, owing to peculiarities in locality or from the union of several patches, they may sometimes present various shapes, although their normal shape is round or oval; the margin or outline of each patch is always sharply defined, and the patches are generally slightly raised above the surface. Very soon after their first appearance the spots become covered with a pearly-white epithelial coat, which may increase greatly, and is shed constantly with ordinary friction: in strumous subjects this scaly covering becomes thicker and of a more yellow hue and more adherent. When the scales are removed from a patch of psoriasis, the remaining surface is of a bright-red color, and a little scraping will raise a thin tenacious epithelial layer, a pellicle or membrane, beneath which the surface is still more red, and very slight scraping of this will abrade the exposed papillæ and cause blood to appear.

Various names are given to the eruption according to the size and appearance of the individual lesions, and for other reasons; in every instance, however, the disease is one and the same, and they are really of little practical value. Thus, when first appearing in minute points, it has the name *psoriasis punctata;* when the minute lesions enlarge so as to represent, as was fancifully supposed, "drops of mortar," the name *psoriasis guttata* is given; still larger patches, supposed to suggest a coin, are named *psoriasis nummularis*, or *discoides;* when it tends to clear in the centre, the name *psoriasis circinata* or *orbicularis* is given; and when extending circles produce irregular-shaped lines by the union of several of them and the clearing up of certain portions, the term *psoriasis gyrata* is employed. Other designations are also sometimes used, such as *psoriasis diffusa* or *universalis,* to indicate a very general eruption, and *psoriasis inveterata,* to express its rebellious character in particular cases. When the crusts are much heaped up, as in strumous subjects, the term *psoriasis rupioides* has been applied; and when more or less blended with eczema, the name *eczematous psoriasis* is used.

There are generally few if any subjective symptoms in connection with the eruption of psoriasis; the spots are commonly seen before they are felt, and there are no constitutional symptoms belonging to the disease. Sometimes, however, the eruption will itch considerably, especially when first developing; and when a large surface is attacked, and the lesions have been torn or the scales removed by very active treatment, there may be considerable burning; fissures also sometimes occur, which may prove very painful.

Diagnosis.—Typical cases of psoriasis, when fully developed, seldom present much difficulty in diagnosis when all points of its clinical history are considered: but when ill defined, or when confined to certain regions, the eruption may occasionally be difficult of recognition. The only eruptions likely to be mistaken for it are *seborrhœa, eczema squamosum, lichen planus, lupus erythematosus, tinea trichophytina,* and the *scaly syphiloderm.*

1. *Seborrhœa.*—On the chest the circular red patches of this eruption often resemble psoriasis closely, but the scales are more greasy, and the eruption would be absent from the extremities: on the scalp seborrhœa presents greasy scales on a pale base, and the edges of the eruption are ill defined, large areas being affected; in psoriasis the scales are still dry and branny, the patches are separate and sharply defined as elsewhere, and the base red.

2. *Eczema Squamosum.*—The patches of this eruption are ill defined, generally larger than those of psoriasis, the scales are more adherent and less abundant; the centre of the patch is apt to be hard and perhaps fissured, and there is commonly a history of more or less moisture and crusting in some patch; the location is more apt to be on the flexor surfaces and there is much more tendency to itching than is exhibited in psoriasis; moreover, the patches never begin with a minute point and develop slowly, as do those of the latter eruption.

3. *Lichen Planus.*—The papules of this eruption are generally grouped, flat, shiny, and depressed in the centre, not scaly, and of a violaceous color; they are more apt to attack the flexures of the wrist, seldom if ever the elbows, and itching is generally a marked feature.

4. *Lupus Erythematosus.*—This rarely occurs in children, and it is only on the face or scalp that its hard, red, irregular patches, with a small amount of horny, adherent scaling, could possibly be mistaken for psoriasis.

5. *Tinea Trichophytina.*—Ringworm of the scalp or body presents

patches spreading peripherally which might be confounded with those of psoriasis. But the eruption is rarely if ever symmetrical, the scales are never abundant or silvery, and the parasite may be readily found in them with the microscope, there being also generally the history of contagion.

6. *Syphilis.*—The papulo-squamous syphiloderm may resemble psoriasis very closely, but almost always a close study will develop some places presenting features very different from this eruption, and very commonly other signs of syphilitic disease can be discovered. The scales of the specific eruption are never so abundant and silvery as in psoriasis, and are far more adherent, and the bases of the lesions far more succulent, and of a darker, coppery red: moreover, the eruption of syphilis will generally be found equally developed, or even more so, on the flexor surfaces, and generally affects the palms and soles as well, which are spared in psoriasis.

Prognosis.—While never tending, so far as is known, to endanger or shorten life, psoriasis is one of the most annoying of all affections, for both the physician and the patient. The prognosis must always be guarded as regards a permanent cure, for while the eruption may often disappear, sometimes very promptly, under treatment and occasionally without, the tendency is very great for the disease to reappear, even after it has been absent months or years. A strong effort should be made, with children especially, to treat it vigorously and persistently at its first appearance, and to endeavor to keep it in abeyance as long as possible, in the hope that with developing age the tendency to it may be outgrown, aided by the proper treatment and the regulation of the health by every available means.

Treatment.—Cases of psoriasis differ very greatly in regard to the treatment required, and some care must be exercised in adopting appropriate measures: the weakly and spare subjects require different handling from the hearty and fat ones, and some skins are infinitely more susceptible than others to the effect of irritating applications. There is no one well-defined line of treatment suitable to every case and every stage of the eruption, and much useless or even painful medication may easily be practised in this disease. No definite statements can be made in regard to the dietary management of psoriasis, but it appears that an excess of meat is harmful, and cases do better where its use is restricted to a considerable degree; but, on the other hand, an excess of sweets and starches is also bad, especially in gouty subjects, while an increase in the use of simple fats aids in the treatment of the disease.

Constitutional treatment should first be directed to rectifying any existing derangements and to raising the tone of health. For this purpose, alkalies are often required, with diuretics and cathartics, followed by iron and bitter tonics; cod-liver oil is exceedingly beneficial in certain cases, and phosphates may also prove very serviceable. Arsenic undoubtedly stands almost alone as a special remedy for psoriasis, and in some instances, when properly pushed, demonstrates its powers in a striking manner, and, as children, as a rule, bear arsenic remarkably well, it may be given freely, when necessary, and even until it acts on the bowels. But arsenic is often contra-indicated by an inflamed condition of the eruption, and it is seldom desirable to give it while the eruption is actively developing. Generally arsenic does better when combined with an alkali or an iron tonic than when given alone.

Local Treatment.—Care must be exercised in ordering local treatment for children, for it is very easy to give remedies which will do harm while they do very little good. Chrysophanic ointment is rarely if ever applicable, nor are the stronger, almost caustic applications generally recommended for this eruption. The following will be found a most serviceable application: ℞ Acidi carbolici, gr. v (or resorcin, gr. x); bism. subnitr., ʒss; unguent. hydrarg. ammon., ʒi–ʒii; ung. aquæ rosæ ad ℥i. M. To be thoroughly rubbed into the affected patches, either alone or after washing with—℞ Acidi salicylici, ℈i–ʒi; spts. vini rectif., ℥i; glycerini, ʒiv; aquæ rosæ ad ℥iv. M. This may prove a little strong, and should be used weaker for delicate skins. Alkaline and starch baths are often very serviceable, and may be given with advantage several times weekly. There are many applications recommended for psoriasis, the principal of which are oil of cade, diluted with oil or in ointments, salicylic acid, three to five per cent. in spirit, naphthol, ten to fifteen per cent. in ointment, etc. But great care must be exercised in using these or any irritating remedies on the skins of children, and sometimes a mild astringent, like the calamine-and-zinc lotion, will serve better than almost anything else.

LICHEN SCROFULOSORUM.

Definition.—A chronic inflammatory eruption, composed of small, moderately-elevated papules, seated around hair-follicles, of a tawny-red color, more or less grouped, slightly desquamating, and without itching, occurring in scrofulous subjects.

History.—While the description of this disease given by Hebra has been mainly relied on in the past, the eruption has been more and more recognized in this country, and is not now thought to be so rare as once was the case. It gives so little trouble that it is apt to be overlooked, and may be found even when the patients or friends are unconscious of or ignore its existence. It is not an eruption of importance, and is chiefly to be recognized for differentiation from syphilis.

Etiology.—Other than the scrofulous condition or diathesis, there is no cause known. It is a disease of young life, cases rarely being seen over thirty years of age; Crocker reports it in a child eleven months, and Neumann in a child four years and a half old: it is said to be more common in males.

Pathology and Pathological History.—The disease consists in a low grade of inflammation and cell-infiltration in and around the hair-follicle and its sebaceous glands, as also in the papillæ around the follicular opening; the exudation takes place from the blood-vessels supplied to the hair-follicles and their sebaceous glands, and when excessive it fills their cavities, pushing off the root-sheath from the wall of the hair-follicle. As the process retrogrades, the cellular deposit is absorbed, and some slight scarring may ensue, although as a rule they leave no trace of their existence.

Symptomatology.—Lichen scrofulosorum comes on with no general symptoms, other than those of a lowered general health, flabby tissues, pale and perhaps coated tongue, with sluggish action of the digestive organs: there are few if any subjective symptoms, it rarely itching at all. Groups of papules form quite suddenly, and continue to be produced from time to time, so that the eruption, if unchecked, may last for years, and the individual papules may remain for months unchanged. The seat of preference of the eruption is on the trunk, but the limbs become affected later. The papules are all small, often not larger than the head of a pin, slightly elevated, of a dusky, livid red, becoming pale later, and capped with a slight epidermal scaling.

Diagnosis.—The eruption might be mistaken for a *papular eczema*, a small *papular syphiloderm*, a *punctate psoriasis*, and *keratosis pilaris*. *Papular eczema* presents more inflammatory lesions, of a brighter red, and is far more itchy than lichen scrofulosorum. The *syphilitic eruption* which most resembles that under consideration occurs early in the secondary period, has larger papules, of a deeper and duller red color, forming more rapidly, and is pretty sure to develop upon the limbs as soon as on the trunk; there will also almost surely be other signs confirmatory of syphilis, such as mucous patches, bone pains, alopecia, etc. A fine *punctate psoriasis*, just developing, may present lesions quite resembling lichen scrofulosorum, but a very brief period will suffice to distinguish them by the increase in size of the psoriatic papules and the formation of the characteristic silvery desquamation. *Keratosis pilaris* appears mainly on the extremities, the papules are harder and more horny, the elevations are minute and grayish, and without anything which could be compared to the little scales or the papules of lichen scrofulosorum.

Prognosis.—This is in the main favorable, although the disease may prove stubborn to treatment.

Treatment.—The prime element is the removal, as far as possible, of the strumous tendency or diathesis which is at the bottom of the difficulty: cod-liver oil seems to be the sheet-anchor in lichen scrofulosorum. Externally the free use of emollients, as vaseline, or cold-cream with a quarter part of lanoline, generally suffices, with free bathing, to remove the eruption.

LICHEN PLANUS.

Some recent authorities have classed the *lichen ruber* of Hebra with the eruption commonly known as *lichen planus*, as originally described by Wilson, applying the term *acuminatus* to the former in distinction from the *planus* of the latter. Inasmuch as the identity of the two affections is by no means proved, and the real lichen ruber of Hebra is exceedingly rare in this country, and very rarely if ever occurs in children, the description in the present article will pertain wholly to what has been called *lichen ruber planus* but is commonly known as *lichen planus* alone.

Definition.—Lichen planus is an inflammatory disease, characterized by the presence of small flat papules of a purplish-red color, shiny, and generally with a slight central depression, discrete or confluent, running a chronic course, and attended with more or less itching.

History.—The eruption commonly begins without any antecedent symptoms, the attention frequently being first drawn to it by the itching. Patients generally appear to be in perfect health, but careful investigation will commonly reveal more or less of ill health, chiefly in the direction of suboxidation of tissue. The eruption is exceedingly chronic in most instances.

Etiology.—No very certain statements can be made in regard to the etiology of this eruption. It cannot be excited by any local means, nor does it follow any well-determined constitutional condition. It occurs about equally in the two sexes, and, though it is most frequent at about middle age, it may appear at any period of life, and is occasionally met with in children.

Pathology and Pathological Histology.—The papules of lichen planus arise from an inflammatory process in the papillæ and upper part of the corium, giving rise to a mass of round-cell infiltration, which may be accompanied by a thickening of the rete, but the corneous layers are generally thinned, especially in the centre. According to both Robinson and Crocker, a sweat-duct is generally found in the centre of the papule, which seems to be the principal cause for the umbilication of the lesion; and the latter observer thinks the sweat-glands must have something to do as "determinants for the starting-point of the process."

Symptomatology.—The most common site for the first appearance of the eruption is about the wrists, especially on the flexor surface, and few cases will fail to present the lesions here some time during their course; it may, however, appear on any portion of the body, but is rare on the face. The papules composing the eruption, when fully developed, are peculiar and very characteristic: they present a flattened appearance, with abrupt, wall-like sides, the surface being glazed or shiny, and with a depression more or less marked in the centre. They may appear quite separate and distinct, but are apt to become congregated together, so that sometimes a patch of considerable size is formed: on such old patches there may be a slight production of scales, but as a rule there is little if any scaling, even until the papules disappear by absorption.

Diagnosis.—The eruption might be mistaken for a *papular eczema*, or for a *papular syphilitic* eruption, and possibly for *erythema papulatum*. The lesions of *eczema* are more acute, are generally acuminate, presenting also vesiculation somewhere, and erythematous patches, or even a moist or crusted surface; there is also more burning and itching than in lichen planus. The small flat *syphilitic papule* of hereditary syphilis sometimes simulates lichen planus closely, but a careful examination will commonly reveal some spots larger, more succulent and evenly glazed, and also of a more dusky red color and not umbilicated; there will also be more or less abundant eruption about the mouth and anus, locations spared by lichen planus; other signs of syphilis may also be readily found, as this eruption occurs at an early period, when the poison is active. The papular form of *erythema multiforme* commonly comes on the backs of the wrists and hands first, the lesions are larger, more rounded, not umbilicated, and with more inflammatory disturbance, and are apt to present somewhat varied appearances, with occasionally vesication.

Prognosis.—Unlike real lichen ruber, lichen planus never endangers life, nor does it interfere greatly with personal comfort, although in some instances the itching will be a really distressing feature. But the eruption is always a tedious one, and, while some cases may be cured in a few weeks, in many instances the eruption will persist, in spite of treatment, for many months.

Treatment.—In children the eruption almost always presents acute and rather congested papules, and the indications are for remedies, internal and external, which allay the vascular excitement and reduce hyperæmia: when this is done, the itching generally ceases and the eruption fades. Mild laxatives and acetate of potassium afford most relief in the acuter forms; later, quinine in free doses is of service, and, when the eruption persists and becomes less active, arsenic, pushed even to full doses, will generally check the eruption. In more acute conditions the calamine-and-zinc lotion with carbolic acid (gr. v–x ad ℥i) yields most benefit; later, carbolized vaseline (gr. x ad ℥i) following alkaline baths generally suffices to remove the eruption.

GANGRENE OF THE SKIN.

History.—Gangrene of the skin in children may occur in the course of or follow erysipelas, scarlatina, measles, and varicella, and also in connection with other skin-affections, as pemphigus and syphilis; or it may develop spontaneously, as far as can be ascertained, without any known cause. The extensive sloughing of the face seen in cancrum oris, or noma, is excluded here, as belonging to gangrenous stomatitis. Gangrene may occur at any age, immediately after birth or later in childhood, about equally in both sexes, and is generally seen in those who are cachectic or otherwise enfeebled, although it sometimes occurs in those in apparent health. It runs a variable course, and attacks all portions of the body. The general symptoms are usually severe, with fever at first and a depressed temperature later in the disease, especially towards a fatal termination.

Etiology.—Little is known in regard to the causation of cases of gangrene, other than a depressed vitality, and some special tendency to localized derangement of circulation, whose ultimate cause is unknown.

Pathology and Pathological Anatomy.—It is quite possible, if not probable, that the gangrene associated with the exanthemata is due to local infection with microbes, favored by the lowered vitality of the patient. In regard to spontaneous gangrene, it is pretty evident that it is dependent upon a spasm of the arterioles, causing local asphyxia, but whether this is of central or reflex and peripheral origin has not been determined with certainty: Hochenegg[1] has demonstrated material changes in the spinal cord in certain cases of symmetrical gangrene.

Symptomatology.—Idiopathic gangrene is generally more or less symmetrical, affecting the fingers or toes, rarely both, the vulva, the scrotum, or other portions, quite suddenly and without apparent cause; it may also appear in a disseminate form, numerous patches occurring in various localities. The part about to be affected becomes of a dull-red or livid color, which grows darker in color as the disease advances: the part feels hard and may be tender on pressure, or the sensibility may be greatly diminished from the first, though there are darting and burning pains, which may be very severe. With this there are irritability, loss of appetite, headache, malaise, and some fever. The disease may be arrested before gangrene sets in, and the parts gradually return to a normal condition. More commonly it progresses, a slough forms, and greater or less destruction of tissue may follow, even involving all the tissues of a limb. The gangrene may be dry or moist, and after the separation of the slough complete healing may take place in a comparatively short time.[2]

Diagnosis.—This is seldom difficult, although it is often impossible to determine the exact nature of the complaint until serious symptoms have set in, which is, however, generally pretty soon after its commencement.

Prognosis.—This is always serious, for, while some cases recover, the disease is commonly fatal: when the gangrenous process is extensive, or when it appears successively on different parts of the body, there is little hope: the most favorable cases are those of limited dry gangrene.

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[1] Hochenegg, Ueber symmetrische Gangrän, etc., Wien, 1886. (This work contains a very complete bibliography of the subject.)

[2] Many peculiar features have been recorded in connection with this subject which cannot even be noted here, and for further consideration reference may be made to Crocker, "Diseases of the Skin," London, 1888, p. 273, and Eustace Smith, "A Practical Treatise on Disease in Children," New York, 1884, p. 166.

Treatment.—Little can be done, beyond the measures suggested by ordinary medical knowledge. The utmost possible support should be given to the system, together with quinine administered with a free hand, and opium in small and frequently-repeated doses. Galvanism has proved of service in the adult, applied either from the spine to the affected part, or with both poles on the latter. Strong nitric acid is recommended to be applied to the affected spots, to arrest the sloughing process, and the resulting ulcerations should be treated on general surgical principles, and with disinfectants.

ECZEMA.

By ARTHUR VAN HARLINGEN, M.D.

Definition and Nature.—Eczema is an inflammatory, acute or chronic, non-contagious disease of the skin, characterized at its commencement by erythema, papules, vesicles, or pustules, or a combination of these lesions, accompanied by more or less infiltration and itching, terminating either in discharge with the formation of crusts or in desquamation. It is eminently a protean disease. At one time it begins as an erythema; later this may become moist and secreting, and finally terminate in a thickened, dry, and desquamative surface. At another time the affection may begin in the form of vesicles or pustules, with swelling or heat. These soon burst, and a red weeping surface results, which is soon coated with bulky crusts from the drying of the liquid gummy discharge. The character of the patch may then suddenly change, and instead of a weeping surface there may exist a dry, scaly, infiltrated, fissured patch of skin, which continues until the disease is removed. Or, again, papules may first appear; these may remain as such throughout their course or may pass into other lesions, or they may be associated sooner or later with vesicles. There is no other disease of the skin in which the lesions undergo such sudden and manifold changes, and every variety may manifest itself in turn upon the same individual. More or less itching is almost always present in eczema. It may vary in degree from the merest titillation to unendurable torture. Sometimes burning takes the place of itching; at other times they occur together. Eczema may be acute, running its course in a few weeks and then permanently disappearing, or it may be chronic and continuous or recurring through years. It may occur in small patches single or multiple, or more rarely covering extensive surfaces. It is *never contagious.*

Etiology.—The etiology of eczema in children, and especially infantile eczema, is by no means thoroughly understood. Some observers are inclined to give great weight to diathetic causes, as scrofula, etc., while others believe that most if not all cases of eczema in infants and young children can be traced to the operation of external irritants.

Prof. James C. White, of Boston, in a paper of great weight and value,[1] draws attention to the external factors in the etiology of eczema which come into play the moment an infant is born into the world. "From its prolonged, placid, subaqueous life it [the infant] emerges into sudden contact with the more stimulating properties of an entirely different element, the atmospheric ether. For the first time its capillaries dilate to their fullest extent under the new conditions of respiration, an independent and intensified circulation, and spasmodic vocalization. So, too, its glandular systems are called upon to adapt themselves to the strange external surroundings,—the sebaceous follicles to modify the character of their secretion, the sweat-glands to perform their functions, probably for the first time.

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[1] Some of the Causes of Infantile Eczema, etc., Boston Medical and Surgical Journal, 1881.

"Moreover, at this critical period the infant makes an abrupt acquaintance with the foreign materials of the outer world. Anointed at once with fats, too often a rancid vegetable oil; then rubbed with a chemical compound, more frequently than otherwise composed of impure constituents and so imperfectly combined that an excess of alkali is at liberty to exercise its caustic action upon the susceptible skin; then plunged into water of varying temperature, and briskly rubbed; and finally received upon a coarse blanket and dried by friction it may be with a coarse towel,—such is often the first treatment the skin receives. Later the dressing: around its abdomen is bound tightly a broad flannel band, between its legs are stuffed thick folds of napkin, and about its lower extremities again the rough contact with the woollen petticoat,—all ingeniously adapted to irritate the skin by overheating, pressure, and rude friction.

"It is not surprising under these circumstances that the skin should resent such irritative surroundings and should within a few days develop a fugitive congestion of greater or less extent, or a mild follicular inflammation which may develop into the more serious and permanent form of eczema."

But other exciting causes are at work. The discharges are often allowed to remain too long unremoved. The irritating fæcal matter and urine kept in contact with the skin by thick folds of napkin can scarcely fail to produce the erythematous condition called intertrigo or chafing, from which to eczema is but a step. Among the poor, neglect in these matters is a common cause of eczema, to which must be added the regurgitation of milk allowed to saturate the clothing about the neck throughout the day and night. Imperfect removal of the smegma at the first washing, and too warm and thick clothing, inducing profuse perspiration, may also be exciting causes of eczema.

To indicate the probability of such causes being at the bottom of most cases of infantile eczema, White tabulates his cases with the view to showing the age at which the disease is most likely to occur. Out of 5000 cases of eczema treated by him at the Massachusetts General Hospital, 1890 occurred in children of ten years of age and under, as shown by the following table:

Within the first year of life	569 cases.
Between 1 and 2 years of age	286 cases.
Between 2 and 3 years of age	280 cases.
Between 3 and 4 years of age	198 cases.
Between 4 and 5 years of age	144 cases.
Between 5 and 6 years of age	118 cases.
Between 6 and 7 years of age	93 cases.
Between 7 and 8 years of age	76 cases.
Between 8 and 9 years of age	66 cases.
Between 9 and 10 years of age	60 cases.

Taking out the operation of the causes directly acting upon the skin from without, above mentioned, and a few other extraneous agencies, the parasitic chiefly, White does not hesitate to say that he knows nothing whatever of the causes of the disease in the remainder. Eczema affects all classes of society alike; it occurs at all seasons of the year; it comes in children of all degrees of health, in the perfectly sound as well as in the feeble, "and," says White, "in equal proportion among bottle babies and those fed at the breast." His observation gives him no justification for believing the various other assigned causes for the disease.

Bulkley[1] takes a somewhat different view of the etiology of eczema from that expressed by White. He does not so rigidly exclude the operation of internal causes. Heredity, in Bulkley's opinion, has little influence in the production of eczema. Vaccination it may be positively asserted cannot cause eczema, though, like any other cutaneous irritant, it may provoke an eruption in one strongly inclined thereto. "While," says Bulkley, "the fact cannot be denied that very many infants with eczema, perhaps the majority, look to be in perfect health, . . . I feel confident in affirming that exceedingly careful medical investigation will always discover something to be corrected besides the disorder of the skin; certain it is that a very rigid investigation and regulation of the diet, mode of life, etc., together with appropriate aid from medicines, accomplishes for these little ones what local treatment has failed to do."

For my own part, I think that both of these distinguished dermatologists are right; and while, with Bulkley, I would urge examination into every possible weak point in diet, hygiene, hereditary tendency, and general nutrition, I would at the same time, with White, enforce the necessity of careful examination into all local circumstances and extreme attention to local treatment. Of course neither of these writers lays any stress upon a specific tendency to eczema, an hereditary or acquired "taint" as in syphilis. Such notions are no longer held by any one who has studied the disease from a scientific stand-point. However, when I come to speak of treatment I shall be found suggesting the internal use of various drugs. Not only shall I recommend laxatives, etc., but also tonics, and in scrofulous cases iodine compounds. Do I then consider eczema a "scrofulous" disease?

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[1] Eczema and its Management, New York, 1881.

By no means. I do not even know what definition should be given to this term. But one thing I feel sure of; that is, that eczema, or at least the predisposition to eczema, is induced by any cause which depraves the general nutrition, and that the various signs which are generally recognized as indicative of the scrofulous tendency go hand in hand with symptoms of impaired nutrition, and point also, when found in connection with eczema, towards a certain plan of treatment which may perhaps be called anti-scrofulous. Dyspepsia, too, is a predisposing cause of eczema, and likewise the anæmia which accompanies and results from mal-assimilation of food. These etiological factors must, I think, be considered in our study of the disease. That eczema is *caused* by teething I do not assert; but that outbreaks or relapses of eczema occur with great frequency during the pressure of teeth upon the gum just previous to their breaking through is a matter of daily observation.

Among older children the local conditions favoring the occurrence and persistence of eczema are those which can be traced to original and inherent vulnerability of the skin. There are persons whose skins, though apparently healthy, are dry and what are called "thin." They seem too soft to resist external irritants. Others have that peculiar congenital failure in development of the skin known as ichthyosis, which markedly predisposes to eczema. I think I can sometimes observe the ichthyotic tendency even in very young infants; but with each month of life it develops more and more, until at from one to two years of age it is often perceptible, while a few years later it is so obvious as to strike even an unpractised eye.

The ichthyotic skin is abnormally dry, rough in some parts of the body, as the elbows and knees, smooth, tight-drawn, and shining in others, as over the nose and cheeks and about the hands. The insides of the hands and fingers in a child of seven to ten who has the ichthyotic skin even to a slight degree will be found smooth, thin, wrinkled almost like the inside of a monkey's paw. The condition is peculiar. It is the one local predisposing cause of eczema in older children which is characteristic. With it often goes asthma, and, as I have observed in some cases, hypertrophy of the mucous membrane of the nasal cavity. I have never been able to satisfy myself as to the connection between these affections, but content myself with noting their concurrence.

Symptomatology.—The varieties of eczema are named according to the lesions which the disease presents at its beginning. These are the *erythematous*, the *vesicular*, the *pustular*, the *papular*, the *red* or *eczema rubrum*, and the *squamous*. All of these may occur in children and infants as well as in adults, but the appearances they present are different, owing to the anatomical and physiological characteristics of the skin during the early weeks, months, or years of life. For this reason I shall here diverge from the general description of eczema, and go on to describe the various forms of the disease as they are encountered in infancy and childhood.

Erythematous eczema shows itself in typical cases as an erythematous state of the skin, of which that chafing so common about the groins and nates is a characteristic example. In fact, the border-line between what is known as simple erythema and erythematous eczema is a very indefinite one. We can say little more than that in one instance we find congestion only, and in the other more or less exudation and infiltration, usually, however, very slight in degree. Erythematous eczema in infants may occur in large or small patches without discharge or moisture. Commonly the patch is covered with fine thin scales of epidermis, and now and then the surface, especially in fat infants where the skin lies in folds, is slightly excoriated. The skin may be bright or dark red; it sometimes has a yellowish tinge. It is not unfrequently mottled. The process may affect a large surface or a small one or may occur in scattered patches. It is often better one day and worse the next, and it may even go away entirely only to return a little later.

The localities affected by this form of eczema in infants are chiefly those where warmth, moisture, and irritative discharge favor and provoke congestion and maceration. Thus, the folds of the buttocks, the groins, and the genitals are usually the first seat of the eruption, which may from these points spread to other contiguous surfaces. The neck, chest, and shoulders are likewise favorite seats of this form of eczema, although it may occur on any part of the cutaneous surface. It is the earliest of all the forms of eczema to appear, and may be encountered within a few days—one may almost say hours—after birth. Of the causes which may produce it, we shall speak under the head of the general etiology of the disease, and of the diagnosis, which, in respect to its possible confusion with syphilis, is a matter of moment, we shall likewise deal under the general head of diagnosis.

Eczema erythematosum may run its course as such, gradually improving when the causes producing it have been done away with, or when it has been relieved by appropriate medication, or it may develop into eczema vesiculosum or eczema rubrum or rarely into eczema squamosum.

This form of eczema, as has been said, is peculiar to infancy; it is much rarer in children of older growth.

Vesicular eczema is one of the commonest forms of the disease both in infants and in older children. It is rare during the first weeks of infancy, but may develop after the third or fourth week, and is the form most commonly met with from the sixth week to the third year of life and even much beyond.

Vesicular eczema commonly begins by a feeling of heat and irritation in the part, which shows a diffused or punctate redness, with itching and burning, and small vesicles soon show themselves, either alone or grouped, or sometimes running together. They are soon filled with a yellowish gummy fluid, and then they ordinarily break and form a crust. Sometimes, however, the vesicles simply dry up without breaking. In more marked cases, new crops of vesicles continue to come out, and, when a considerable surface is covered, the quantity of fluid poured out is quite large and the underclothing or dressings are saturated. When the secretion dries, it is very sticky and tenacious; and this is characteristic of this form of eczema.

Typical vesicular eczema, as described, is not so common as the more complex varieties where the lesions are multiform,—papules, papulo-vesicles, vesicles, pustules, and other lesions being found in conjunction. The two chief characteristics of this form of eczema, wherever found, are the itching and the gummy secretion, leaving a yellow stain upon the linen. As found upon the face and scalp of infants, this form of eczema constitutes the affection popularly known as *milk-crust, scalled head, tooth-rash,* or *moist tetter.*

Pustular eczema, in some of its forms termed *impetiginous eczema,* is likewise met with both in infants and in older children. It is very much the same in its original appearance as vesicular eczema, only that the lesions assume the form of pustules rather than of vesicles. There is usually less heat and itching. A strict line cannot always be drawn between the two forms, for they are apt to run into each other, and may coexist upon the same subject and even in the same patch. The scalp and face are favorite seats of pustular eczema, and it is apt to occur in children who are badly nourished or scrofulous. The same causes which would bring out a vesicular eczema in a tolerably healthy child will give rise to the pustular form in the weakly or poorly nourished. This is shown in the production of boils, so common in the latter case.

Papular eczema is not often encountered in the infant, and is unusual even in older children. It is a form of eczema much more frequently found in the adult than in the young. It appears in the form of small round or acuminate papules, varying in size from a small to a large pin's head. In color the lesions are bright or dusky red, sometimes violaceous. They may be discrete, or may run together, forming large patches, and these are often hard and infiltrated. Now and then they become abraded and moist, forming one variety of eczema rubrum. Papular eczema is apt to occur on the arms, trunk, and thighs, especially the flexor surfaces. In children this form of eczema rarely if ever reaches the extent that it does in adults, but the lesions, like those of papular eczema, are the seat very often of violent itching.

Eczema rubrum is rather a secondary than a primary form of disease, always resulting from a previous condition of eczema erythematosum or eczema vesiculosum, more rarely from eczema pustulosum or eczema papulosum. In eczema rubrum the surface of the skin is inflamed and infiltrated, red, moist, and weeping; occasionally the diseased area is more or less covered with yellowish or brownish crusts, often completely overspreading the part. When these crusts are not removed, but are allowed to adhere, secretion or rather exudation meanwhile goes on underneath, and the appearance presented is that of a thick rough yellowish, greenish, or brownish crust, cracked here and there, and oozing the gummy fluid noted above under vesicular eczema.

Eczema rubrum may occur in any part of the body. In infants, however, the scalp and cheeks are the ordinary seat of the disease. A more superficial form of the disease, with less discharge and little or no crusting, is observed especially about the genitals of male children and sometimes about the buttocks and thighs.

In older children, when eczema rubrum occurs, which it does much less frequently than in infants, the arms and legs, as in adults, are apt to be attacked. The affection in these latter cases is not often so extensive or severe as it is among adults or among young infants.

Squamous eczema, like eczema rubrum, is rather a secondary than a primary condition. It results from a previous erythematous, vesicular, pustular, or papular eczema. Usually in children it follows erythematous eczema. When typical, it shows itself in the form of variously sized and shaped reddish patches, which are dry and more or less scaly. The skin is usually slightly infiltrated or thickened, but this thickening is rarely present to a perceptible degree in infants and young children. The condition is commonly ephemeral, but may become chronic.

Having now described the symptoms of each form of eczema, let us observe how these appearances are grouped to form a picture of the disease as met with in practice. And, first, the eczema of infants at the breast and of very young children.

An infant born healthy and with a pure unspotted skin, save only for the remains of the pre-natal smegma on the scalp over the fontanels, which has been left there by the too cautious mother or nurse fearful of injury to those tender parts, begins to develop a slight redness and discharge, with crusting, around these patches. In a day or two papulo-vesicles, vesicles, and vesico-pustules appear in increasing numbers in the neighborhood of the original patches. These rapidly coalesce, the weeping and crusting increase, and in a very short time the entire scalp is a mass of scales and crusts and the seat of violent itching. The infant shows signs of distress by moving its head from side to side, and, when lying down, rubs the occiput constantly against the pillow until it is often nearly denuded of hair, the hairs being broken off short near the surface by constant attrition.

If allowed to run on without attention, the eczema may spread beyond the scalp down behind the ears, when the skin soon becomes red, glazed, and weeping. Fissures form behind the ears, which in extreme cases seem so deep that it appears as if the ear must be on the point of dropping off. Sometimes the weeping is so profuse that dried serum and crusts attach themselves to the lobe of the ear, while the discharge runs down over them and hangs and drops like stalactites.

The face may become the seat of eczema in connection with the disease of the scalp, and we here usually find the eruption spreading from three centres,—the forehead and the middle of each cheek. The lesions are very similar to those on the scalp, rapidly-forming vesicles, crusts, raw and cracked surface with considerable weeping. The itching is as severe here as on the scalp, and infants learn to rub and tear the cheeks at a very early age, so that excoriations, blood-crusts, and drops of fluid blood go to make up the picture.

Occasionally, in connection with this form of infantile eczema, the eruption breaks out about the neck and shoulders, when it is apt to take on the erythematous form. The amount of vesiculation, weeping, crusting, etc., is much less in this locality.

The course of this form of eczema is apt to be chronic, and, unless treated with great vigor, it may go on from bad to worse, lasting for months, dying away for a time and then with the approach of teething lighting up again into a fresh exacerbation. It is the form of eczema which is perhaps most frequently encountered during the first two or three years of infantile existence.

Various degrees of the affection are encountered, from that just described down to the mildest form, which is characterized by little more than a redness with slight infiltration in the cheeks, and an occasional eruption of small scattered vesicles or vesico-pustules. Or there may be a slight scaling or crusting in the scalp, and no more.

Another typical form of eczematous eruption is that which finds its origin about the anus, buttocks, and genitals and spreads from these points down the thighs and up over the back and abdomen. I cannot better convey the idea of this eruption than by describing a case which came under my care some time ago.

A lady brought to bed of her second child was attended in the country by an ignorant and inefficient monthly nurse. She suffered from peritonitis after confinement, and the nurse in her anxiety for the mother neglected the proper care of the infant, who, moreover, was fed with more or less carefully prepared artificial nourishment at various irregular intervals and was probably not kept very scrupulously clean.

At the end of a month eczema had developed to a marked degree, and I was called in to examine and prescribe. I found the infant in a pitiable condition. Sleeplessness, insufficient and improper food, colic, and continued suffering from itching and burning had affected the little patient's nutrition; he was thin almost to emaciation, the dry skin hung in wrinkles and folds about his limbs, and his wizened face presented a pitiable expression. On taking off the clothing and making a thorough examination, the head, face, chest, arms, and legs below the knees were found free from disease, but the buttocks, thighs, back, and genitals were the seat of a severe eruption of erythematous and red eczema. The integument was slightly infiltrated, dry, red, shining, and tense. About the folds of the groins, testicles, and penis, and around the anus, were moist fissures and cracks. The frequent acrid discharges from the bowels, coming in contact with the fissures about the anus, gave rise to acute pain, and each passage caused the infant to shriek with anguish. There was in this case no inherited tendency to scrofula or eczema. The eczema was evidently the result of local causes combined with insufficient and improper nourishment, and removal of the cause, with appropriate local applications, resulted in a speedy cure.

A form of eczema sometimes met with in very young infants, but more frequently in older nurslings and young children, is characterized by the appearance of circumscribed patches of disease, usually red, infiltrated, slightly moist patches of eczema rubrum, about the arms and legs, and occasionally accompanied by vesicular eczema of the hands and feet. This form of eczema is more chronic and intractable than those above described. At times it breaks out anew with the eruption of teeth, at other times it continues without marked change, unless active treatment be instituted, and may linger on during the first three or four years of the child's life. Unlike the other forms, it seems connected with some ill-defined condition of the general system or some inborn defect of the skin, as will be mentioned a little later in the description of eczema as it occurs in older children.

Although the varieties of eczema above described form well-defined types, such as will be recognized by any one who has seen many cases of eczema in children, it must not be supposed that all cases will conform to one or another variety. It not unfrequently occurs that two or even all of these forms are encountered in a single case or at one period or another of the disease. A recognition of the various forms will, however, I am inclined to think, aid in the investigation of a given case and in ascertaining what etiological factors enter into its occurrence.

Among older children who have been subject to eczema in infancy, and even among those who have not previously shown signs of the disease, eczema may break out at any age, but not often with such severity as in early infancy. The eruption may here take on any of its forms. We may have pustular eczema of the scalp (to be carefully differentiated, it should be said, from pediculosis capillitii), vesicular eczema about the face, hands, body, or limbs, and eczema rubrum behind or within the ears. In older children, too, we are more apt to encounter the papular variety of the disease. Eczema here does not differ very greatly from the disease as met with in adults, only, however, owing to the greater delicacy and vulnerability of the skin, the eruption may occur more suddenly and yield more readily to treatment. Besides this, I have observed that pustular eczema is more apt to occur in children than in adults, and also that eczema rubrum in large areas over the lower limbs, so often encountered in older persons, is rarely if ever met with in children.

Diagnosis.—The diagnosis of eczema in children does not usually offer much difficulty. When, in scrofulous infants, a pustular eczema occurs upon the face, and especially about the mouth and nostrils, and when, in addition, there is a certain amount of nasal catarrh, often a chronic condition among poor and neglected infants, syphilis might be suspected. The syphilitic pustules, however, are much larger, more severe-looking, and are apt to rest upon a red, hard base of new cell-formation. Not unfrequently furunculoid lesions, which, however, are not, strictly speaking, furuncles, but in reality gummata, are met with in connection with the syphilitic eruption. Moreover, the "snuffles" of hereditary syphilis is a greenish purulent discharge tending to dry and clog in the nasal passages, while the nasal discharge in chronic eczema is mucus or at most muco-pus.

When, in infants, the disease is confined to the buttocks and adjacent parts, it is sometimes difficult to make the diagnosis between eczema and syphilis. Commonly, however, if a close examination is made, some infiltrated and cracked papules will be found about the anal orifice in syphilis, or some patch of induration more deep than that met with in eczema, or some characteristic patch of disease will be met with elsewhere, notably in the form of fissures at the edge of the lips or crusts in the nostrils.

Eczema is liable to be confounded with pediculosis capillitii, more commonly in very young infants, although indeed lice may be found at any age. The diagnosis may usually be made, first, by observing the locality of the disease. Eczema may occur all over the scalp; pediculosis affects the occipital region, the crusts and scales with pustules being found only there and extending down over the back of the neck. In the second place, the insects themselves may often be found, and their nits attached to the hairs almost always.

Eczema in infants and young children may be confounded with scabies. Here, too, the distribution of the parasitic disease is regular and uniform. The hands, anterior fold of the axillæ, abdomen, buttocks, thighs, and feet are the favorite seats of the pustules. Moreover, in infants suffering from scabies, the peculiar and characteristic burrow of the insect can almost always be made out, especially on the hands. In eczema, patches of disease occur here and there; in scabies the eruption is discrete. Though both affections are markedly pruritic, scabies itches very much worse at night, while the itching of eczema is tolerably constant.

Tinea circinata and tinea tonsurans may be mistaken for eczema, and *vice versa*. When ringworm occurs upon the body, however, in children, it usually grows so luxuriantly as very soon to betray its characteristic features of annular shape, fine scales, and regular progression. When ringworm occurs on the scalp, the diagnosis is at times more difficult, but the presence of the short broken-off hairs of the sharply-defined ringworm-patch, which presents a nibbled appearance, is so characteristic that an attentive examination will always reveal which of the two diseases is present.

Treatment.—The treatment of the eczema of infants must often be both local and general, but the local treatment is of the most importance, and success or failure will in many cases depend upon the manner in which it is carried out. The disease in young infants is usually acute. First erythematous eczema, then papular rapidly running into vesicular, then, as the effect of scratching and rubbing, pustular and weeping red eczema result. A certain degree of infiltration accompanies most cases after a short duration. All these forms are accompanied by severe and intense itching, and the scratching and rubbing induced tend greatly to aggravate the disease and promote its extension. How young infants can bear the strain on the nervous system induced by such attacks of itching and the attendant sleeplessness, which drive adults to frenzy, is more than I can comprehend. But they do endure it, and sometimes even flourish under it, and it is often as much to give rest to the parents and attendants as to relieve the patient that the physician is called in. "A child," says Dr. White, "may lay waste the strength and health of a household by the care it demands through months and months of nights and days, and remain at last its only healthy representative in all respects save its skin, retaining its nutrition, plumpness, vigor throughout. The health of those in charge of it becomes in fact eventually the chief object in view in the care of the baby."

How, under these circumstances, cases of severe eczema in infants can be allowed to run on month after month without local treatment under the advice of a physician, passes my imagination. But such instances are met with, and under the plea that it would be dangerous to cure such an eruption for fear of "driving it in." I had thought that such excuses were no longer made except by unprincipled quacks, but while writing this article I came across a clinical lecture addressed to medical students in which the "Professor" actually adduced two cases of fatal convulsions in eczematous infants to prove the danger of external applications to dry up the eruption! Hebra, with an experience of twenty-five thousand cases of eczema, declared that he had never seen any injury supervene upon the cure of eczema; and such is the universal testimony of those who have had the most experience in this disease. In fact, were it not for such instances of the almost immortal longevity of error, it would seem that no allusion need be made to the subject.

Before giving examples of the applications most likely to prove useful in the peculiar cases of infantile eczema we are considering, I wish to emphasize, by quoting again from Dr. White's paper, the importance of a thorough application of whatever is used. In infantile eczema even more than in other skin-diseases a mere prescription with general directions to apply, rub on, etc., will prove perfectly futile.

So long as there is eczema and hyperæmia there will be itching, so long as there is itching there will be scratching, so long as there is scratching there will be no chance for the excoriated skin to heal. As it is impossible that the infant can be constantly held day and night by nurses and attendants, some form of mechanical restraint must be applied, and this is what is recommended by Dr. White:

"A skull-cap is to be made of fine old cotton or linen cloth so as closely to fit the calvarium; a mask of the same material is then shaped to the face, with exactly-placed apertures for the eyes, nose, and mouth, and with slits for the ears. It is to be gathered in somewhat beneath the chin, and made long enough to lap some two inches at the back of the head. This in mild cases will prove to be a sufficient protection against the efforts of the infant to get at the irritated skin with its hands, and a shield against the damage inflicted by rubbing the inflamed parts against every opposing surface which offers. It is sometimes sufficient that such a mask and cap should be worn only when the child is sleepy, the only time when it is generally left unwatched; but such partial use is permissible only in the mildest grades of the disease. But the protection from irritation afforded by the mask is only one of its important duties: it may also be made to take a valuable part in the direct treatment of the disease. Of course its use will never interfere with the application of any other class of remedies to the skin, but it may be smeared with ointment, and, adjusted tightly, form an impermeable coating to the inflamed skin. It may be worn in this way for twenty-four hours without change, or removed at shorter intervals for the application of such remedies as the case demands. The nose and ears should protrude through their appropriate openings to assist in retaining the mask in position, which should be tightly stitched or pinned with fine safety-pins at the back of the head. But generally additional means must be employed against mischief, as the hands of a strong infant are capable of doing injury both to the mask and the skin beneath during the paroxysms of itching, or of developing the disease upon the neck or other parts. It is generally, therefore, best in all but the mildest cases of the affection, even when confined to the head, to use a sort of strait-jacket in addition to the mask. A hole is to be cut in the end of a small pillow-case large enough to allow the child's head to pass through. This is to be drawn down over the body and arms. The back and front surfaces are then to be stitched together between the arms and body by a long darning-needle, from the axillæ down to the ends of the fingers, thus confining the arms in closed sleeves to the sides. The same result may perhaps be more readily accomplished by the use of several safety-pins in place of the stitches, by which the jacket may be more readily taken off when necessary. The pillow-case is then to be fastened together by the pins between the legs from front to back, so that the arms cannot possibly be brought up to the head. This lower fastening can of course be removed without trouble as often as it is necessary to change the napkin. We have thus rendered the hands completely harmless. The mask and jacket are of course resisted by the little patient at first, but in a day or two are worn, when adjusted, without a struggle. The jacket should be worn day and night, and while removed for the application of other dressings or during the bath the hands are not for a moment to be left unheld by an additional attendant. . . . It is astonishing what results are often accomplished within twenty-four or forty-eight hours by the mask and jacket. . . . Not until the skin is completely restored to its normal condition, or at least until all signs of the inflammatory state and of pruritus have disappeared, are these mechanical means of restraint to be relaxed.

"When the disease is more extensively distributed, covering the arms and legs or the whole surface as well as the head, . . . it often becomes necessary to confine the feet and legs as well as the upper extremities, to prevent their constant friction against each other. The same method of pinning through the pillow-case from front to back should be employed, following the inner line of the legs from the crotch to the feet, while they are kept some distance apart. If the outer edge of such trousers be then fastened to the bed or cushion on which the child is seated, the legs can neither be drawn up nor approximated to any dangerous contiguity.

"Whatever ointments are required may be applied either on the inside of the cap and mask, or on cloths, and the pillow-case drawn over the whole dressing."

I have given Dr. White's system of restraint at some length because I think it a very important adjuvant to our means of treatment. When I describe, as I am about to do, the various local applications which may be made in infantile eczema, it must be understood that these are to be applied, in severe cases and when practicable, on the plan given above.

Parents will sometimes rebel against the disfigurement of the child by these forms of dressing, and tender-hearted persons may consider a method which prevents the infant from scratching himself when he itches to be reprehensibly cruel. But what seems to be cruelty at first will prove in the long run the greatest kindness, and the rapid relief given must outweigh sentimental considerations.

When we come to consider the remedies employed in the local treatment of the eczema of infants, we are appalled at the innumerable formulæ with which we are presented, particularly in the medical press. Lotions, powders, pastes, ointments, of every possible sort and in all imaginable combinations, are recommended as specifics in many cases, and too often without regard to the circumstances of locality, nature, or stage of the eruption. What will suit admirably one stage or variety of eczema will be injurious in another, and we must select our remedies with reference to the character of the lesions in each individual case.

Before specifying the particular remedies which will be most useful, one or two principles of treatment may be mentioned. In the first place, an acute eruption should generally be treated with soothing remedies. But when a fresh exacerbation of a long-standing eruption occurs, we may sometimes employ more stimulating applications at once. Instances of this will be given below. In the second place, vesicular eruptions should not ordinarily be treated with soap and water. The crusts which form over vesicles, if there is not much itching, should not be washed off or picked off unless it is certain that decomposition is taking place underneath. They may be softened and gently removed by soothing cataplasms. When, however, there is purulent exudation, the resulting crusts and other débris should usually be removed as soon as possible, because decomposition rapidly sets in, with the production of irritating compounds. In the third place, when itching is severe and when this symptom is evidently aggravated by the rapid formation of vesicles, these may be broken open to give exit to the secretion and relieve the itching. When there is itching with infiltration, stimulating remedies come into play.

It must also be remembered that the infant's skin absorbs more readily than that of older persons, and consequently a certain caution must be observed in the employment of mercurial and lead preparations, which should not be employed over too large a surface, for fear of producing toxic symptoms.

In very acute eczema lotions are often of value. One of the best of these is the *lotio nigra*, or black-wash. This is made, as is known, of calomel and lime-water, and consists of a light precipitate of black oxide of mercury with a large proportion of supernatant solution of chloride of calcium. Which of the ingredients is most active I cannot say, but it is usually best to shake the mixture well before applying, to get the virtues of the solution and precipitate. I do not think that the black oxide of mercury can be absorbed by the skin, and therefore this preparation can be used freely even over large surfaces. It may be dabbed on with a rag, or bits of soft rag may be wet with it and employed as an evaporating application; the rags may be allowed to become nearly dry and should then be wet again. If allowed to become perfectly dry, they are apt to stick to the skin and to cause irritation and pain when removed. If covered with an impermeable covering, they are converted into cataplasms and may do injury by macerating the skin. Sometimes, after dabbing on the black-wash for some moments, it may be followed by some mild ointment, as the oxide-of-zinc ointment, pure or diluted with vaseline. This plan may be used at night or when for any reason the eruption cannot soon again be dressed. The ointment may be applied with the finger or on rags and bound on with bandages, or in severe cases with the harness above described.

If for any reason it is desirable to use ointments alone, those which are most soothing are first to be chosen, unless the eruption is decidedly chronic in character. The following formulæ represent the best qualities of soothing preparations. The first is the *unguentum diachylon of Hebra*,—not the mess which is now unhappily officinal, and which will usually be dispensed by the apothecary as a tough and stringy mass or as a slimy fluid.[1]

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[1] Hebra's formula is as follows:

℞ Olei olivæ opt., f℥ xv;
Pulv. lithargyri, ℥ iii ʒ vi;
Aquæ, q. s.
Coque. Fiat unguent.

The oil is to be mixed with a pint of water and heated, by means of a steam-bath, to boiling, the finely-powdered litharge being sifted in and stirred continually; the boiling is to be kept up until the minute particles of litharge have entirely disappeared. During the cooking process a few more ounces of water are to be added from time to time, so that, when completed, water still remains in the vessel. The mixture is to be stirred until cool. The ointment is difficult to prepare and requires skilful manipulation. When properly

I think unguentum diachylon, properly made, is one of the most soothing and sedative of all ointments, and it will agree when ointment of any kind can be borne, though there are some cases in which no unguent will agree with the inflamed skin and when lotions alone can be employed.

Next to diachylon ointment in my estimation comes the oleate-of-bismuth ointment of McCall Anderson, which I introduced to the profession in this country some years ago, giving it the name of the distinguished dermatologist who devised it. This is composed as follows:

℞ Pulv. bismuthi oxidi, ʒi;
Acidi oleici, ℥i;
Ceræ albæ, ʒiii;
Vaselini, ʒix;
Olei rosæ, ♏iii.
M.

This when well made is, in the pharmaceutical sense of the word, "elegant." It resembles butter in appearance and color, and when skilfully perfumed is a most agreeable preparation.

Other soothing ointments may be made of carbonate of zinc or subnitrate of bismuth, in the proportion of a drachm to the ounce of cucumber ointment.

It is useless to multiply formulæ further in this direction. Properly applied, one of those above given will provide the necessary protection for the parts, with a sedative and slightly astringent effect.

When the eruption has reached the chronic stage or when it presents itself in a subacute condition, somewhat more stimulating applications may be made. The following combination is useful:

℞ Pulv. hydrarg. chlor. mite, gr. v–x;
Ung. zinci oxidi, ℥i.
M.

An equal quantity of carbolic acid may be added as an antipruritic. The mercurial is not likely to produce any constitutional effect when used about the face and scalp in the strength above given. I have never seen mercurialism from its use.

When the eruption has reached the subacute or chronic stage, or in carefully-selected cases even earlier, tar in several shapes will often be found useful. This is particularly the case in those instances of the disease where the eruption is dry, red, shining, and infiltrated. Especially in small

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made, it should be of a light yellowish color and of the consistence of butter. To insure a good article it is essential that the very best olive oil and the finest litharge be employed. The physician should examine each lot as made up when this is possible, and he should in all cases decline to employ any ointment which has been on hand over a week. Unguentum diachylon is probably more apt to be ill made or decomposed when dispensed than any other, and it behooves the physician to look carefully after his prescription if he desires to avoid a possible catastrophe to his reputation.

patches of red eczema of the cheeks will this drug be found useful. I use the following formulæ with satisfaction :

℞ Picis liquidæ, ʒi;
Adipis, ℥i.

Or

℞ Picis liquidæ, ʒi;
Ung. zinci oxidi, ℥i.

Sulphur may be used in combination with tar, and very often with the happiest results :

℞ Sulphur. præcipitat.,
Picis liquidæ, āā, ʒss;
Ung. zinci oxidi, ℥i.
M.

This preparation may be used in cases where not only the head is attacked but where the arms and legs or body also show numerous infiltrated patches of disease. It, as well as the other tarry preparations, should be used in small quantity and rubbed thoroughly into the skin, not merely applied upon the surface like the more soothing preparations.

Infantile eczema occurring in the erythematous form in the axillæ, about the groins, genitals, anus, buttocks, thighs, etc., requires somewhat different local treatment from that just described as suitable for the scalp, face, etc. The cause of this form of eczema is usually local irritation; either excessive sweating from too warm clothing or the irritation of urine and acid fermenting fæces.

One of the first things to do in eczema of this kind is to abate the cause, whether it be sweating or acrid discharges. Something will be said of the internal treatment of such cases farther on, but here I may remark that in mild cases simply coating the surface from time to time with vaseline, especially about the anus, for the purpose of protecting the skin, will often cure the eruption. In this form of eczema—eczema intertrigo in many cases and little more—powders and lotions are effective. Powdered starch may be used in the mildest cases, but if there is much moisture or discharge we cannot employ this, because it quickly becomes converted into a paste, and in a short time this paste decomposes, grows sour, and acts as an irritant. Lycopodium is a good powder; better perhaps is finely levigated kaolin, subnitrate of bismuth, or carbonate of zinc.

When there is evidently much itching and burning, but no discharge, the following combined powder gives great relief. It should not be applied on raw surfaces :

℞ Pulv. camphoræ, ʒi;
Pulv. amyli,
Pulv. zinci oxidi, āā, ℥ss.
M.

These powders may be dusted on, or may be rubbed abundantly with the woolly side of a piece of patent lint and bound upon the skin.

In some cases lotions are to be preferred to ointments. The best of these is black-wash, described above. Dilute lead-water may also be employed. The lotions should be applied on rags, and, unless there is considerable discharge, the rags can be allowed to dry between each fresh application.

Eczema about the buttocks, genitals, etc., will sometimes bear the application of tarry preparations, especially the tar and sulphur ointment above given. Extreme cleanliness is essential, especially in this form of eczema, while in that about the head and face soap-and-water sometimes does harm.

When the eczema is acute and extensive, covering large areas or scattered over the body and limbs, warm medicated baths are often of the greatest service in connection with other forms of treatment. Two ounces of carbonate of sodium dissolved in about fifteen gallons of water with half a pint of clear starch stirred through the water is a good formula. When the infant or child is taken out of the bath, any appropriate application of those mentioned above can be used.

Older children suffering from eczema may be treated in the same manner as adults, and will usually bear the use of the same local applications. When the occurrence of eczema seems to be favored by an ichthyotic condition of the skin, daily warm baths with soap, followed by general inunctions with some bland oleaginous material, aid in the preservation of the skin from eczematous attacks. When these occur, they are to be treated in the same way as the eczema of adults, for which reference may be made to well-known text-books on dermatology.

The general treatment of infantile eczema, though important, has nothing specific about it. It is directed towards removing all sources of irritation, internal and external, which may excite the inflammation of the skin, and improving the general nutrition when this is impaired.

In early infantile eczema digestive disturbances are very commonly at the bottom of the disease, while in the eczema of older children some fault of nutrition must be combated. It would be easy to give a list of digestives, antacids, anti-fermentive remedies, tonics, etc., but these are familiar to all, and their employment, with the indications for it, will be found pointed out at greater length and more thoroughly in other parts of this work.

Prognosis.—The prognosis of eczema in children is favorable. Most cases of infantile eczema can be cured in periods varying from a few weeks to months, if the source of irritation can be removed. When the eczema depends upon some general defect of the skin, as ichthyosis, the prognosis must be more guarded. In some cases relapses may occur at intervals during the whole period of childhood to adolescence, in spite of all treatment.

PURPURA.

By ARTHUR VAN HARLINGEN, M.D.

Definition.—Purpura is an affection of the skin characterized by the development of variously sized and shaped, smooth, reddish or purplish hemorrhagic patches, which may or may not be elevated above the surface, and which do not disappear under pressure.

History.—The affection is one which has attracted attention in modern times only. We find but scanty and vague mention of hemorrhages into the skin in the writings of classic and mediæval authors. Werlhof in the early part of the eighteenth century first described purpura with sufficient detail to induce a general recognition of the affection as a morbid entity. The severe forms of the disease have since been called in Germany "morbus maculosus Werlhofii," but neither the importance of Werlhof's work in this direction nor the clinical histories he gives are sufficient to substantiate his claim to give a name to the disease, nor even to establish its existence as a type. The name purpura is and should remain the true designation of the affection.

Of late years our knowledge of the clinical history of purpura has been greatly increased by numerous reports on the subject with histories of cases. A great number of varieties and subdivisions of the disease have been described, with the result hitherto of rather confusing the subject than simplifying it. Some knowledge has been gained as to the etiology of the disease, and many theories have been put forth regarding its pathology; but little real advance in our knowledge of this aspect of the affection has been gained.

Etiology.—The chief predisposing causes to purpura, so far as these are known, appear to be derangements of the digestive organs, hæmophilia, and the condition known as scurvy, induced by improper food and damp and unwholesome dwellings. Sudden changes in the circulation, as in the so-called "purpura neonatorum," are said to bring on purpuric extravasations. Want of support to the vessels due to the relaxation of the tissues after long illness, etc., may induce the same condition. Diseases of the viscera, of the spleen, liver, kidney, and cardio-vascular system, diseases of the nervous system, specific fevers, acute septicæmia, pyæmia, and syphilis, and also the ingestion of certain drugs, as iodine, quinine, salicylic acid, copaiba, belladonna, ergot of rye, chloral, chloroform- and benzoic-acid-inhalations, phosphorus, mercury, and the mineral acids,—all this long list of causes may be mentioned as giving rise to hemorrhagic exudation. But only those first named are cited as producing idiopathic purpura, and those causes are so vague that it would almost seem better to confess our ignorance and to say that in most cases of idiopathic purpura no predisposing cause can be adduced.

Purpura is more common in females than in males, and is decidedly more common among the young, as the following table from Gintrac will show:

FREQUENCY OF OCCURRENCE OF PURPURA AT VARIOUS AGES.

In 3 cases the age was	2, 3, and 8 days.
" 51 " " "	1 to 10 years.
" 46 " " "	10 to 20 "
" 30 " " "	20 to 30 "
" 22 " " "	30 to 40 "
" 16 " " "	40 to 50 "
" 16 " " "	50 to 60 "
" 6 " " "	60 to 70 "

One hundred cases, therefore, were observed before the age of twenty years, and only ninety in all the subsequent five decennial periods.

Pathology.—Of the pathology of purpura something more is known than of its etiology, but not very much. The immediate cause of the extravasation of blood is probably to be found either in some change in the quality of the blood itself, some alteration in the structure of the vessels, or some fault of innervation.

The blood-changes found to exist in connection with purpura are—1, greater or less diminution in the number of red corpuscles and in the quantity of solid materials; 2, inconstant variations in the proportion of fibrin (diminution in purpura hæmorrhagica and infectious diseases, increase in purpura simplex and scurvy); 3, increase in the proportion of white corpuscles; 4, change of form in the red corpuscles; and, 5, the presence of abnormal elements, as embryonal elements and bacteria. The latter are known to act mechanically in plugging up the capillaries.

As to the vascular changes in purpura, fatty or amyloid degeneration in the walls of the capillaries has been brought forward as one cause of hemorrhagic extravasation. Few cases, however, have been adduced in support of this view. Inflammation of the smaller vessels (endarteritis) has been observed in several cases of purpura; whether, however, this has been the cause or the result of the extravasation has not as yet been made clear. In some cases examined, vascular dilatation with stasis has been observed. The fact that purpura so often occurs in connection with other stasis-affections of the skin, such as the exanthemata, seems to point to this condition of congestion and stasis as playing an important part in the hemorrhagic eruption. Disturbance of the capillary circulation and augmentation of blood-tension are two elements which should be considered in any future inquiry into the pathology of purpura.

As to the question how these disturbances of the capillary circulation originate, it appears that in many cases they seem to arise from some vasomotor disturbance of innervation. The fact that ecchymoses have been produced by injuries inflicted upon the spinal cord, and the observation long ago made by Weir Mitchell that severe painful points developed in the skin are in some cases followed by the appearance of ecchymoses, seem to add weight to the theory of a nervous origin of the disease.

Symptomatology.—The symptomatology of purpura as this occurs in children is not markedly different from that met with in adults, except as regards a certain class of cases to be described farther on. It is my opinion, however, that the severer forms of purpura are more common among children.

Setting aside the symptomatic forms of purpura, which will also be touched upon under the various diseases in which they are liable to occur, we shall here consider only those forms of the disease which are properly called idiopathic.

These are customarily divided into *purpura simplex* and *purpura hæmorrhagica*. Although an arbitrary division, this is a convenient one, and we shall first consider the symptoms presented by purpura simplex.

In *purpura simplex* the first symptom is usually the eruption of a greater or less number of pin-point- to pea-sized ecchymoses, generally unaccompanied by any subjective symptom whatever. The mother or attendant of a child who appears at the time to be in the enjoyment of perfect health may observe in dressing or undressing him a few minute bluish or purplish spots on the upper or lower limbs. Next day a few more appear, and the physician is called in. At first the history given may be that of perfect health, but close questioning will frequently elicit the fact that there has been some lassitude, want of appetite, and malaise for the previous day or two. The spots on examination are found to be of various size, from pin-head to split-pea size, or in rare cases as large as a cent. Some of the smaller ones are of a bright or dark red color, and if only a few hours old may be slightly raised above the surrounding skin and may partly disappear under pressure of the finger. The older lesions, however, are bluish or purplish, not raised above the level of the skin, and do not at all disappear under pressure. The spots are rounded in outline and sharply defined from the neighboring skin, which preserves its normal color.

The locality chiefly affected by the eruption is the surface of the legs, to which indeed it may be confined. The feet, both the instep and the hollow of the sole, the thighs, buttocks, scrotum, and prepuce, are the localities next most frequently affected. Of the upper portion of the body the forearms are most commonly invaded. The trunk and face are generally spared. In addition to the lesions above described, one or two patches of ecchymosis resembling bruises may at times be observed.

The mucous membranes are not attacked in purpura simplex. The gums are healthy: there is no fetor or swelling about the teeth. Occasionally there is slight epistaxis. The eruption gives rise to no subjective symptoms. There is no pain, itching, or heat in the disease-patches. Children often play about as usual. The pulse is natural; the digestion, the respiration, the excretions, and the nervous functions all seem normal.

The spots have scarcely appeared in the skin before they begin to undergo evolution. The color becomes darker, then yellowish or greenish yellow, and then begins to fade, usually from the circumference towards the centre. Occasionally, however, the involution begins at the centre and works outward, giving the older lesions a ringed look. A given macule commonly goes through the various degrees of evolution in about two weeks,—depending, however, on the patient's health. As the eruption appears by successive outbreaks of new lesions, it is usual in examining a case in full career to observe the lesions in every stage of evolution, from the pin-head-sized, red, slightly-elevated lesions of a few hours, to the dusky or fading yellowish-brown patches about to disappear by absorption.

The duration of purpura simplex is variable. If the patient remains in bed there may be only a few successive crops of lesions. But if the patient is about on his feet, and especially if he stands or walks for hours together, new crops come out daily, and the disease may continue indefinitely. Of course in children this state of things does not often obtain, and, so far as my personal experience goes, this form of purpura does not last usually more than a few weeks to a month.

Purpura hæmorrhagica, or the severer form of purpura, may occur either in persons enjoying apparently the best health, where it usually manifests itself suddenly and, so to speak, in a sthenic form, or in persons of depressed vigor and poor health in a slow and chronic form.

As has been remarked in speaking of the general etiology of purpura, the predisposing causes are not accurately known. Cold weather and cold and damp dwellings seem to be the only predisposing conditions which are generally accepted as inducing purpura hæmorrhagica. Perhaps the existence of a tendency to hæmophilia may be included as predisposing to purpuric eruptions; but many "bleeders" do not have purpura. Recently masses of bacteria have been found in the ecchymotic patches, and the occurrence of a number of cases simultaneously has given rise to the term "infectious purpura" to describe this variety.

The severer forms of purpura are usually ushered in by some precursory symptoms,—a feeling of weight in the head, pain in the limbs, and general malaise. At times the first symptoms are similar to those which mark the onset of a severe exanthematic fever. The skin is hot, the face flushed and the expression excited, the eyes bright and injected, the pulse full, hard, and frequent. The eruption when it appears is first seen upon the lower extremities, and later comes out on the trunk, the arms, and even the face. The lesions are similar to those seen in purpura simplex, but, in addition to the small patches called petechiæ, larger areas, known as ecchymoses, are also observed, and the lesions are more numerous, more sudden and general in appearance, and last longer. The symptom usually considered to mark the distinction between purpura simplex and purpura hæmorrhagica is the occurrence of hemorrhages into the mucous membranes in the latter. These are not always present even in severe and fatal cases, but when they occur add to the gravity of the prognosis. Occasionally the effusion of blood in purpura hæmorrhagica is so superficial or so intense as to give rise to the formation of blebs containing blood or bloody serum.

The duration of the individual lesions in purpura hæmorrhagica is longer than in purpura simplex. Once fully developed, the color remains unaltered about a week; then it begins to change to brown, then to yellow, and then to fade. Commonly the entire evolution of the lesion exceeds over a month or six weeks.

The hemorrhage in purpura hæmorrhagica may occur not only into the skin, but also wherever there is a mucous membrane. Epistaxis is most frequent in children. Hæmatemesis is less common, and bleeding from about the pharynx and tonsils is rare. When hæmatemesis occurs, there is usually some pain in the left hypochondrium, with splenic enlargement. When the stools contain blood, this is usually dark and grumous, rarely pure and bright red. Hæmaturia is marked by discoloration with clots, the proportion of blood being at times so considerable that the urinary odor is lost. Menorrhagia sometimes occurs. Hemorrhages from the lungs and intestines are among the commoner symptoms among children as among adults. Cerebral hemorrhage may also occur among children.

In connection with purpura, especially of the severer forms, œdema, circumscribed or general, may occur and form a marked symptom.

Somewhat different in appearance from the forms of purpura just described, and perhaps between them with respect to severity, are the varieties known as "purpura urticans" and "purpura rheumatica." In the first of these, itching is a marked symptom of the disease, and occasionally urticarious lesions precede and accompany the purpura. Sometimes a small urticarial wheal appears, and shortly after a hemorrhagic spot appears in the centre of the lesion, which gradually sinks to a level with the skin, leaving the simple purpuric stain behind it. In purpura rheumatica, rheumatoid pains about the joints constitute a very marked accompaniment. By some observers this affection has been considered a purpura due to or accompanying rheumatism. Others consider the joint-pains to be simply the result of hemorrhagic effusions in the serous membrane of the joints. I believe that the results of post-mortem examinations have supported the latter view, purpuric effusions having been found in the synovial membranes.

A variety of the so-called purpura rheumatica is that not unfrequently met with when, in addition to the rheumatoid pains, there is violent epigastric pain, colic, and pain in the back, followed not unfrequently by bloody vomiting and bloody stools. Albuminuria is likewise met with. In rare cases the large effusions of blood under the skin give rise to gangrene; and, as this may happen at various points, on the face as well as on the limbs, death, or survival with hideous cicatricial deformity, has been recorded.

A form of purpura called "fulminant" (*foudroyant*) by the French has recently been described. The following case reported by Hervé is typical of this variety:

> An infant, three months of age, after twenty-four hours of restlessness and malaise, was examined, and found pale, with rapid respiration and pulse and slight mucous râles in the chest. No vomiting or diarrhœa. The legs, thighs, and abdomen showed a dozen hemorrhagic spots the size of a ten-cent piece. A second examination four or five hours later showed a marked increase in the number and size of the ecchymoses, which now appeared over the mouth and face. The pulse was imperceptible, the infant extremely agitated and weeping, while still taking the breast with avidity. There were no hemorrhages in the mucous membranes. The ecchymotic patches continued to spread, so that the lower limbs looked as if dyed in wine-lees and were quite œdematous and cold. Ecchymotic patches appeared at all points, and the patient died in about ten hours after the first appearance of the ecchymoses.

This frightful form of the disease is, fortunately, very rare: not more than seven or eight cases have been reported, to my knowledge.

Diagnosis.—The diagnosis of purpura is not usually difficult. No other disease is characterized by the appearance of hemorrhagic patches which do not disappear under pressure with the finger, and which are without some definite local or general cause to account for them. In children, flea- and bug-bites present in their later stages precisely the appearance of the petechiæ of purpura. Almost invariably, however, the central puncture of the parasite can be distinguished. Scurvy rarely occurs without such circumstances of bad hygiene and improper diet as will at once suggest the cause, and, in addition, the general prostration, swelling of the gums, loose teeth, and deep infiltrations in the subcutaneous tissues will serve to differentiate the two conditions. Hæmophilia does not ordinarily occur in young children. Severe puncture, loss of a tooth, or some accident followed by profuse bleeding, which never occurs in idiopathic purpura, marks the first outbreak of the hemorrhagic diathesis. It is important to distinguish between idiopathic and symptomatic purpura, particularly that due to the ingestion of drugs. Among the drugs above mentioned, under etiology, as liable to produce purpuric eruptions in persons having that idiosyncrasy, quinine and iodide of potassium are most likely to give rise to such outbreaks. Even the most minute doses have been known to cause an eruption of ecchymotic spots; and at least one fatal case has been recorded.

The prognosis of purpura varies greatly between that of the benign and almost trifling purpura simplex and the almost certainly fatal infectious and "*foudroyant*" varieties. If the eruption occurs in an infant it is more serious than in an older child, all things being equal. If the lesions are small and scattered, if they come out a few at a time, if the little patient's general health continues fair or good, the prognosis is favorable. The variety known as purpura rheumatica, even where, in addition to the pain and swelling of the joints, severe epigastric pain, colic, and constipation followed by hæmatemesis and bloody stools supervene, usually ends favorably. On the other hand, where large areas become ecchymosed, especially where the face is attacked and gangrene threatens, the prognosis is much more serious. Where the mucous membranes are affected, where there is bleeding from the mouth, and still more where there is epistaxis, the prognosis becomes grave. A rise in temperature forming the "pyretic" varieties of purpura is of grave import, and in proportion to the suddenness of onset and severity of the fever. The "infectious" forms of purpura are usually fatal.

The prognosis of symptomatic purpura will of course depend upon the character of the chief affection. Purpura medicamentosa usually gets better when the drug is withdrawn; but, as noted above, the possibility of a fatal termination must be considered.

Treatment.—The treatment of purpura must depend somewhat upon the patient's general physical condition and surroundings. Fresh, airy habitation, good food, tonics and stimulants, must often be at once prescribed. It is of the utmost importance that the patient should be kept quiet in bed. The room should be cool, and the diet should be nourishing. A pure milk diet, in children as in older persons, is usually the best. In some cases laxatives are required, and preferably castor oil. This may be employed when there is constipation, even when the stools contain blood, especially if this is coagulated and altered. Ice may be given in some instances.

Among more specific medicaments may be mentioned dilute sulphuric acid, belladonna, arsenic, and quinine. It is better to leave the administration of iron to the later stages of the disease, or subsequently to combat the anæmia following. Turpentine is of great value in many cases. Ergot in the form of fluid extract, or ergotin hypodermically,[1] is of great use in some forms of purpura.

Quinine should be given in antiperiodic doses in cases where malarial influence is suspected. In a case reported by a French writer, purpura rheumatica with agonizing colic was relieved, after the vain use of large doses of opiates, by a full dose of quinine.

Locally, sponging with cold water, astringents, as tannic acid, alum, and vinegar, with local applications of tincture of cinchona and tincture of myrrh or of rhatany to the gums when required, are customary forms of treatment.

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[1] Minich, of Philadelphia, in a child seven years of age suffering from severe purpura hæmorrhagica gave one grain of Bonjean's ergotin hypodermically every four hours until three doses had been given, with excellent result.

HYPERTROPHIES AND ATROPHIES.

By J. E. GRAHAM, M.D.

ICHTHYOSIS.

Synonymes.—Xeroderma, Ichthyosis vera, Ichthyosis congenita; German, Fischschuppenkrankheit; French, Ichthyose.

Definition.—Ichthyosis is a disease of the skin marked by the formation of white masses of epidermis which peel off like thin paper, or of green, brown, or black masses firmly fixed to the skin and separated from one another by deep furrows and lines. It affects usually the whole integument, is congenital, and of a decidedly chronic character.

History.—The disease has been frequently mentioned by the older writers. Avicenna first described it under the head of Albarras nigra. During the Middle Ages it was frequently noticed by physicians, and, owing to its striking appearance, received many fanciful names, such as leontiasis, hystricismus, etc. A celebrated case, Edward Lambert, born in Ireland in 1710, suffered from the disease in its worst form. He was the father of a family all of whom were affected by ichthyosis. This patient with two of his sons made a tour through England, Germany, and France. He was described by Tilesius, a physician of Leipsic, under the name of Stachelschwein-Mensch ("porcupine-man").

Willan was the first author who gave an accurate and comprehensive description of ichthyosis. Of late years, owing to more minute microscopical investigation, the true nature of the disease is better understood.

Clinical History.—I shall describe the disease principally as it appears in children. It will, however, be necessary to give a brief account of its whole course. Although ichthyosis is frequently spoken of as a congenital disease, it is never found in children at birth, and does not usually appear until after the age of two years. The writer has recently seen a child suffering from ichthyosis in whom the disease appeared on the third or fourth day after birth. It first develops in the mildest form, which has been called a pityriasis, then it increases in intensity to form the ichthyosis simplex, and in some cases it goes on to the most aggravated condition, the ichthyosis hystrix or cornea.

For purposes of description the disease has been divided into two varieties,—ichthyosis simplex and ichthyosis hystrix. These, however, merge one into the other, and, as has just been stated, the two varieties may occur in the same patient at different periods of life.

Ichthyosis Simplex.—This variety has also been described under the term xeroderma. In it there is a defective action of the sebaceous glands and sweat-glands, and an exfoliation of the epidermis in the form of branny scales. In a more pronounced form the scales may be small, or they may assume the form of small plates which are separated by well-marked lines. This latter condition frequently gives the appearance of a fish-skin, and has given rise to the term fish-skin disease. If the scales are not removed by frequent bathing or some emollient application, they accumulate and form layers of considerable thickness. The skin may then present a grayish-white, glistening appearance, or it may be yellow, green, or brown in color. In all forms, even when the disease is slightly developed, the skin presents a dull dirty surface, as if it had not been washed for some time.

In many cases of xeroderma the epidermal scales accumulate on the edges of the axillæ and over the elbows and knees in polygonal warty-like masses which are separated from one another by deep fissures. The diseased condition when fully developed extends over the whole surface of the body except the flexor surfaces of the joints, the genitals, the palms of the hands, the soles of the feet, and the face. In some rare cases, however, it has been found over some of these excepted parts also. The parts most severely affected in ichthyosis simplex are the lower extremities, except the feet.

Ichthyosis Hystrix.—This is a more severe form of the disease, and is often found in its greatest intensity in localized patches. In this condition the epidermis, instead of exfoliating, is retained in the form of thick layers. This retention is due to its capability of being longer nourished. In time, however, the crust or layer loosens from the corium and drops off, in some cases leaving the corium quite bare, and in others leaving it still covered by a more or less thick layer. The masses of epidermis when fully developed either present large wart-like excrescences separated by deep fissures, or occur in the form of long ridges which are also separated by fissures. In different patients these growths appear in different regions, sometimes on the arms alone, and sometimes over the back. Occasionally, however, they are widely diffused over the body. Often the wart-like masses are more or less accumulated and form the porcupine variety. As before stated, the disease appears in childhood at about the second year. It then rapidly develops into that particular form in which it will continue throughout the remainder of life. The usual form found in children is that of ichthyosis simplex. Some cases of the most severe variety have, however, been reported. As a rule, in ichthyotic patients the scalp and hair are dry, and the latter very brittle. The skin of the hands and feet is wrinkled, and there is marked coldness of the extremities. In a few cases the presence of a severe form of some one of the exanthemata has effected a temporary or permanent cure of ichthyosis. Hebra mentions the case of a girl in whom a severe attack of measles caused the disappearance of a previously existing ichthyosis simplex, and another in whom an attack of variola produced a permanent cure. In the latter case pocks occurred only on parts not affected by the ichthyosis, but the whole epidermis exfoliated and the disease never returned.

The affection occurs equally in males and females. It is worse in winter than in summer. It is not unusual to find that ichthyotic patients suffer severely from asthma. They, as a rule, perspire slightly.

Morbid Anatomy.—Upon examining a section of the skin, the epidermal layer is seen to be much thickened, and to be made up of cells bound together by a structureless mass which is not found in the normal epidermis. The discoloration of the masses is due to the incorporation of fat and particles of dirt which adhere to the diseased surface. The corium is thickened and infiltrated. The papillæ are more or less enlarged from hypertrophy of connective tissue and the presence of exudation-corpuscles. A chemical analysis of the ichthyotic masses has revealed some interesting facts. There is an increased quantity of lime salts. Marchand has found silicic acid in considerable quantity. Schlossberger has found silica and oxide of iron.

Etiology.—That form of the disease which comes on in childhood and gradually develops until adult life and then remains throughout life must be looked upon as a congenital disease. The diseased condition has been born with the individual.

The more localized form, according to Hebra, is frequently acquired. Evidence of the hereditary nature of ichthyosis is easily found. There are, however, cases where only one member of the family has been attacked, and no trace of hereditary taint discovered. It occurs in all climates, and in all circumstances of life. Both sexes suffer equally.

Diagnosis.—The peculiar and well-marked character of the skin, together with its history of extreme chronicity, renders the diagnosis of ichthyosis a comparatively easy matter. The thickening of the skin, the large scales with well-marked lines separating them, the wart-like excrescences or ridges separated by furrows which pass deep down to the corium, are very characteristic of this disease. Then its chronic and congenital character will also assist in making a diagnosis.

Treatment.—Internal treatment is of little avail. Cod-liver oil has been found of benefit in some cases. The other alterative remedies—iodide of potassium, arsenic, etc.—are of no use whatever.

For the mitigation of the condition of those affected by the severe form of ichthyosis we must depend entirely upon local treatment. We cannot hope to cure the affection, but we can in many cases give such aid as to make life more comfortable. In children the severe form does not, as a rule, exist, and the treatment is therefore not so necessary as in adults.

The main object of the external management is to soften and get rid of the epidermal masses and at the same time to make the skin more soft and pliable. "Hebra accomplished this by rubbing the patient twice a day with soft soap for from six to twelve days, and placing him naked between blankets till the epidermis began to peel off in large lamellæ. Then the patient had a bath daily for one or two hours or, if possible, for a longer time, and anointed the skin with oil or an emollient salve. By this plan we can cause an ichthyosis to disappear to a great extent; but after a longer or a shorter time the epidermal covering will be reproduced in its former thickness." The emollient application may be made of lanoline or glycerin mixed with two or three parts of cold cream. Glycerin may be combined with oleate of bismuth. The author has found equal parts of vaseline and glycerin of starch of very great use. Duhring recommends the following formula:

℞ Adipis benzoati, ℥i;
Glycerini, ♏xl;
Ung. petrolei, ℥ss.
Sig. Apply daily after washing.

There is no remedy which will prevent the return of the epidermal masses. The local treatment must therefore be repeated as often as it is found necessary.

Prognosis.—Ichthyosis is an incurable disease. As a rule, however, the patient's health is not otherwise injured. Nor does life seem to be shortened by it. The functions of the internal organs appear, as a rule, to be unaffected by it.

SCLERODERMA.

Definition.—Scleroderma is a chronic affection of the skin marked by a localized or general, more or less diffuse, indurated, stiffened or hide-bound condition of the skin. It is usually accompanied by pigmentation, and the patches are often partially anæsthetic.

History.—This disease was first described by Alibert. Recently it has received a good deal of attention from several authors.

Clinical History.—It would seem to occur in two different forms,—one of a more or less acute character which may last for a few months, and another of a very chronic nature. The latter variety is the most frequent. In the acute form the induration is very diffuse. It generally begins in the neck, and gradually extends down over the trunk and extremities, and upwards over the face. The skin does not change much in color, becoming in some cases a little paler. It becomes tense, and an impression of marble is given on touching the affected parts. When, for instance, the neck and face are affected, the parts become rigid as if the intermuscular spaces had been filled with a dense, hard, wax-like substance. The countenance is immovable and looks perfectly smooth. Often the mouth is so fixed that it can scarcely be opened. Two cases of this form have come under the author's observation. Both had suffered from pneumonia previous to the onset of the scleroderma, and both were girls about the age of twenty. This form lasts from six months to a year, when the induration gradually passes away, leaving the skin in a perfectly healthy condition. The chronic variety, which seems to be by far the most frequent, pursues quite a different course. It begins by a more or less pronounced induration, diffuse in character, which is usually first noticed about the neck, but may appear on any part of the body. Often the induration is preceded by a distinct pigmentation. Attention is sometimes first drawn to the part by the stiffness experienced by the patient, and sometimes by the presence of slight febrile disturbance. The disease usually spreads slowly, so that in some cases after many months the whole body may become affected. When fully developed, the skin is tense, hard, and resisting. It is more or less darkly pigmented, and in some cases is partially anæsthetic. The pigmentation is often regularly distributed, so as to give the appearance of freckles. The integument seems to be bound down tightly to the subjacent parts, which are hard and inelastic.

The temperature of the indurated patches is lowered, as shown by the surface thermometer. The difference between it and that of the normal skin is usually from one to one and a half degrees. Generally the affected skin is dry, indicating a cessation of the function of both sebaceous glands and sweat-glands. This is, however, not always the case. The diseased skin does not lose much if any of its vitality, as is shown by the course which the exanthemata take when they attack a scleroderma patient. Intense irritation or inflammation will produce an ulcer more readily in an indurated than in a healthy part.

In some cases the mucous membranes of the tongue, gums, and mouth are affected. Sometimes the sclerosis appears in bands and sometimes in patches. The sense of taste is not usually affected. In scleroderma there are very few constitutional symptoms. Patients generally enjoy fair health, and are discomforted only by the condition of the affected parts. The intercurrent affections, such as tuberculosis, etc., are not the result of the sclerodermic condition. Rheumatic and neuralgic pains are frequently present. This chronic variety may terminate in either of two ways. In some cases, after the affection has lasted months or years, it may end in resolution, the parts resuming their normal functions. Such cases are on the border-land between the acute and the chronic form. Most frequently, however, the indurated patches undergo more or less atrophy. The skin, which was previously thick and hard, may now become thin and parchment-like. At the same time atrophy of the subjacent parts may take place, so that the whole patch may become firmly attached to the bony parts beneath. The skin loses its glossy aspect, and becomes brown and wrinkled. Very often the disease is found in its different stages in the same individual. It is in this latter condition that scleroderma resembles morphœa. In fact, some of the atrophic patches seem identical with morphœa.

Diagnosis.—In a few cases it is extremely difficult to distinguish between this affection and morphœa. In the majority, however, the diagnosis is easily made. Scleroderma affects larger areas and is more diffused than morphœa. The line of demarcation is not so plainly marked.

In scleroderma, also, there is more hardness and rigidity, whereas in morphœa the subjacent parts and even the patch itself may be soft and pliable.

Morphœa is frequently symmetrical and follows the course of nerve-trunks. Scleroderma is unsymmetrical and more diffuse.

In many cases scleroderma comes on much more rapidly than morphœa.

From other affections of the skin scleroderma is easily diagnosed, as the signs are of a definite and positive character.

Etiology.—Scleroderma occurs more frequently in females than in males. According to Van Harlingen's statistics, in twenty-eight cases twenty were females and eight were males. It may occur at any age. As a rule, however, children are seldom affected by it. In a large number of cases rheumatism has preceded or accompanied the disease, or else there has been a family history of rheumatism. For this reason, it is not unusual to find organic affection of the heart present in sclerodermic subjects. Exposure to cold and shock to the nervous system have been set down as causes.

Treatment.—Cases of scleroderma are not frequent even in adults, and no well-defined line of treatment has been laid down. Tonics and alteratives, such as potassium iodide, cod-liver oil, iron, quinine, and strychnine, have been recommended. Gentle frictions with olive oil and the use of the galvanic current have been found beneficial in some cases. The main object of treatment is to improve the general health. This may be accomplished by change of surroundings, the administration of remedies to improve the condition of the digestive system, and the use of plain, easily-digested, strengthening food.

Prognosis.—Recovery takes place in a certain proportion of cases, but in many the condition remains throughout life. Patients are often weakened by contraction and deformity, and are more liable to be carried off by intercurrent diseases.

SCLEREMA NEONATORUM.

Synonymes.—Scleroma neonatorum, Scleroderma neonatorum, Algor progressivus; German, Sclerem der Neugeborenen, Greisenhaftigkeit der Kinder; French, Algidité progressive, Décrépitude infantile.

Definition.—Sclerema neonatorum is a disease which attacks children immediately or soon after birth, and is distinguished by a peculiar, œdematous, corpse-like hardening of the skin. It is accompanied by steadily progressive weakness of the constitution, and nearly always terminates fatally.

History.—The disease was first fully described by Choussier in the beginning of the present century. It was afterwards treated of by Gilbert and Alibert. Full accounts of the affection have since appeared in many text-books on diseases of children as well as in works on dermatology.

Clinical History.—Sclerema may be present when the child is born, or it may appear shortly afterwards. Very often the first circumstance which attracts attention is the unusual coldness of the extremities. The peculiar hardened condition appears in patches on the legs or thighs, and then gradually spreads over the body. The indurated parts often present an intensely red, glossy appearance, often a livid-red or purplish, and sometimes even a brownish color. Occasionally intense pallor is noticed. The skin seems to be stretched over the parts, and the epidermis has a very smooth and sometimes a fissured appearance. As the patches increase in size they unite, so as to produce a general hardening and stiffening of the lower extremities. While this is going on, the child exhibits symptoms of increasing marasmus. The temperature falls two or three degrees each day. The parts have a hard resisting feel, but at the same time they pit on pressure. The sensation on touching them is similar to that produced by a corpse in rigor mortis. The disease then gradually spreads upwards, appearing on the arms, the face, and the greater part of the trunk. The child is then unable to move. The breathing is feeble and shallow, and the patient sinks in two or three days. The face when severely affected presents a peculiar appearance. The parts are hard and immobile. The mouth is partly closed, and the lips are stiff. The wrinkles and furrows in the epidermis give the appearance of an old person. The immobility of the lips prevents the child from taking nourishment, a circumstance which increases the tendency to a fatal result. This takes place often in a few days. If life is further prolonged to two or three weeks, fatal complications, such as bronchitis, pneumonia, peritonitis, are apt to ensue. In a few cases there is a temporary improvement in the condition, during which the temperature rises and some lessening in the size of the patches occurs. In still rarer cases gradual and complete recovery takes place.

Morbid Anatomy.—The indurated portions of the integument do not change much after death; even the color, in place of diminishing, becomes often more intense. When cut into, a yellowish fluid escapes, causing the part to become softer. A dense "stearine-like" deposit is found in the subcutaneous areolar tissue. According to Kaposi, there is no marked increase in the connective tissue of the part. The post-mortem changes, therefore, are of a much more negative character than one might expect from such a pronounced pathological condition. It might be well to state here that Löschner and Jenks have found in some cases an hypertrophied condition of the corium.

Etiology.—This disease is frequently found in premature children. It seems to occur in all conditions of life, a circumstance which has given rise to various opinions as to its causation. There is no doubt, however, that the capillary circulation in the peripheral parts is much involved. Whether this is a primary or a secondary condition has not been well established. It is also a matter of doubt whether the peculiar pathological state of the internal organs, a state which is found in many cases and which is frequently the cause of a fatal issue, is primary or secondary to the peripheral condition. The following diseases have been found to precede sclerema, and may have been etiological factors in producing it,—viz., chronic intestinal catarrh, ulceration of the mucous membrane of the intestine, atelectasis pulmonum, pleuro-pneumonia, chronic bronchial catarrh, malformation of the heart, patency of the fœtal orifices of the heart and great vessels, meningeal apoplexy, hydrocephalus, etc.

According to Kaposi, an hereditary feeble condition, bad nursing, and bad food may also predispose to sclerema. In some cases the disease has been arrested by an improved hygienic treatment of the child. Syphilis also appears sometimes to cause the disease. It has frequently been found in syphilitic children.

Pathology.—As the cause is a matter of uncertainty, the pathology is also obscure. It is probable that through the vaso-motor system the capillaries are first involved, and the other morbid conditions follow as a result.

Diagnosis.—The marked character of the lesion renders the diagnosis a comparatively easy matter. The hardened and œdematous condition of the skin, the peculiar color, and the singular corpse-like feel are found only in this disease. Then, in addition, the cold extremities, the poor circulation, and the feeble breathing are all marked features of this affection. The only disease at all resembling it is the scleroderma of adults, and this runs an entirely different course.

Prognosis.—These cases nearly always terminate fatally after a few days' illness. They may, however, last two or three weeks. A few cases have been known to recover.

Treatment.—The principal object in treatment is to improve the general health. This is best accomplished by improving as much as possible the surrounding hygienic conditions. If the child is not nursing, or if the milk does not agree with it, a healthy wet-nurse should be procured. The circulation should be stimulated by gentle cutaneous frictions, together with the application of external warmth. If intestinal inflammation be present, measures should be taken to remove it. When these efforts are persevered in, a successful issue occasionally follows.

Antisyphilitic remedies should be employed if there is any taint of syphilis present.

ELEPHANTIASIS.

Synonymes.—Barbadoes leg, Bucnemia tropica, Pachydermia spargosis, Elephantiasis Arabum.

Definition.—Elephantiasis is a chronic hypertrophic disease of the skin, which usually attacks the inferior extremities, but may also affect the penis and scrotum in the male and the labia in the female. It commences in the connective tissue and spreads from it to surrounding structures. It is accompanied by subacute and recurrent inflammations of the blood-vessels and the lymphatic ducts, as well as of the neighboring glands.

The disease is found in all parts of the world, but most frequently in tropical regions.

Clinical History.—Whether the leg or scrotum is attacked, the disease is ushered in by an erysipelatous inflammation of the part, accompanied by fever. The parts are swollen, red, and tender, and when the inflammation subsides there remains a permanent enlargement of the part. Frequently, after the disease has existed for some time, abscesses result from the recurrent inflammation. From these pus continues to ooze after the active process subsides. The outlets of pus are frequently marked by wart-like excrescences, which form one of the marked signs of the disease. As a result of these frequent attacks, the parts become enormously hypertrophied. The weight of the leg or scrotum may be so great as to interfere with walking.

Elephantiasis does not occur until after puberty; hence any more lengthened description would be out of place here. The brief account given will, however, serve to introduce a subject which is of considerable interest in connection with the diseases of children.

Hebra, in his manual, calls attention to a third form of elephantiasis which he terms, after Virchow and Hecker, elephantiasis telangiectodes or lymphangiectodes. Some authors, perhaps correctly, treat of this affection as a new growth, and separate it entirely from elephantiasis. It, however, resembles the latter disease sufficiently to be classed along with it.

This condition is always congenital, and has been described under various names. Dr. Busey, of Washington, has recorded a number of cases.

It sometimes remains during life in the same state as at birth, but often it develops so as to produce great deformity. In two cases which I had the opportunity of observing, the disease began in the leg. The affected limb became larger and longer than the sound one, and presented in places a rough nodular surface, produced by the presence of soft round tumors. These were of smooth surface, and made up principally of enlarged vessels. There was also an hypertrophy of all the connective tissues. Even the bones on the affected side were longer and thicker than on the other.

The skin is also hypertrophied, and exists sometimes in immense loose folds. This general hypertrophic condition gives the leg an appearance very similar to that of elephantiasis Arabum. In many cases, owing to hypertrophy of the papillæ, the skin has a soft velvety feel, and presents a roughened appearance somewhat similar to that of the normal integument when viewed under a magnifying-glass. In some cases the color is normal, but there is often deep pigmentation, as in true elephantiasis, and for this reason it is described by Hebra as a variety of that disease.

Elephantiasis telangiectodes may appear in any part of the body, and some rare cases have been reported in which the whole surface was affected.

Pathology.—It is probable that a dilatation of the lymphatics, and in some cases of the blood-vessels, is the first abnormal condition present. Hypertrophy of the subcutaneous connective tissue and of the corium follows. The enlargement of the papillæ produces a peculiar soft sponge-like condition which is often found.

Treatment.—The treatment of the affection is altogether surgical. In some cases, where the disease is localized, it is often possible to remove the enlarged vessels by the electro-cautery and thus prevent the spread of the disease. It may also be removed sometimes after the hypertrophy has become quite extensive.

Patients who suffer from congenital elephantiasis in whom a limb is affected throughout cannot be benefited by surgical treatment, unless it is thought desirable to amputate the diseased member.

Prognosis.—Many of these cases pursue an unfavorable course. There is in the majority deformity more or less pronounced. Very often this deformity prevents the natural movements of the limb and becomes burdensome to the patient. When such diseases as erysipelous eczema or other acute inflammations attack the limb, they run a very unfavorable course. Gangrene sometimes results from the low vitality and from the occlusion of a leading blood-vessel.

MORPHŒA.

Much difference of opinion has been expressed by dermatologists as to the relation which this disease bears to scleroderma adultorum. It has been demonstrated that cases of diffused scleroderma have also exhibited patches of morphœa; but a number of cases of the latter affection have been observed in which the diffused hardening was not present. It is better, then, to consider it as a distinct form of disease.

Morphœa manifests itself in the form of patches, which may be round, oval, or elongated. In its early development the patch presents a whitish or grayish-white appearance, is somewhat harder than the surrounding skin, and is frequently a little depressed. Sometimes there is no perceptible degree of hardness, and sometimes it has a tough, leathery feel. At first, and for some months after its commencement, the patch presents a smooth, shining surface. It then frequently becomes wrinkled, having more the appearance of parchment.

The lesions may appear on any part of the body, but are found most frequently on the extremities and on the face, neck, and chest. They are occasionally found along the course of nerves. There are usually few subjective symptoms, and, were it not for the deformity sometimes produced, the disease would be of little moment. Itching and even pain have been present in some cases. The patches are often found quite anæsthetic, so that the skin may be pierced by a pin without pain.

The patches vary both in size and in shape. On the same patient some occur not larger than a large pin-head, and others, again, as large as the palm of the hand. Occasionally they assume a narrow linear appearance.

The course pursued also varies much. The patches often remain stationary for months, when they quite disappear, leaving the skin in a healthy condition. They may run a comparatively short course and disappear, only to reappear in other parts of the body. In other cases, again, they remain, producing a permanent deformity. If the disease occur in the neighborhood of a joint, it will make it stiff and useless, and when on the face it may also produce marked disfigurement.

The disease has a general tendency to recover, notwithstanding its usual chronic course.

Morbid Anatomy.—Crocker, who has made a careful microscopical examination of sections of affected skin, gave the following description: disintegration of the deep layers of the epidermis, atrophy of the papillæ, and infiltration of cells around the sebaceous glands, hair-follicles, and vessels; in its later stages these cells become developed into connective tissue, and atrophy of the glands, follicles, and vessels speedily follows. Thrombosis of the superficial plexus of vessels has also been noticed.

There is evidently an intimate connection between the nervous system and the development of morphœa. The latter is frequently found along the course of nerves, and the disease is often found in those who suffer from trophic nerve-lesions, such as alopecia areata and canities.

The disease is identical with that described by Addison under the head of true keloid and by Dr. Tagge as Addison's keloid. Its nervous origin is supported by Wilson, Hutchinson, Crocker, and Duhring.

Diagnosis.—Morphœa differs from the diffused scleroderma adultorum in many points. It is more circumscribed, and the lesions are of a deeper character and are followed by cicatrization and contraction. In scleroderma the hardness is more general and the lesions are not so likely to form cicatrices. The latter, again, often runs a comparatively short course,—that is, not longer than two or three months. This never occurs in morphœa. Morphœa also resembles the white patches found in macular leprosy. This resemblance is so close that Erasmus Wilson considered morphœa to be a symptom of leprosy which still lingered in the Anglo-Saxon race. No doubt in both cases the lesions are the result of nerve-affection; but in leprosy the nerve-infiltration is produced by the bacillus lepræ, whereas the nature of the lesion in morphœa has not yet been discovered. There is no difficulty, however, in distinguishing between a case of morphœa and one of leprosy, as in the latter other symptoms of the disease will present themselves. A more difficult diagnosis to make is that between morphœa and vitiligo. In the latter the pigmentary layer alone is affected, and the texture of the skin is quite normal. There is also no anæsthesia present.

Etiology.—Morphœa may occur at any age, and is frequently found in children. It occurs more frequently in females than in males. The strong and robust suffer equally with the weak and debilitated.

Prognosis.—The chronic character of the affection has been already dwelt upon. In the most severe cases the prognosis is not entirely hopeless, as in some improvement, partial or complete, takes place after a considerable length of time. In mild cases a more rapid change is noticed in the diseased patches.

Treatment.—As this is essentially a nervous affection, our efforts should be directed towards the improvement of the general health of the patient, and more particularly to that of the nervous system. Such remedies as cod-liver oil, iron, and arsenic have been found of value. The latter, in order to be of service, must be given in moderate doses for months. The use of the galvanic current when persistently applied is sometimes of benefit.

HYPERTROPHIES AND ATROPHIES.

(CONTINUED.)

By HENRY W. STELWAGON, M.D.[1]

LENTIGO.

Lentigo, or freckle, consists of a localized increase of pigment-matter in the skin, appearing as pin-head- to pea-sized, rounded or irregularly-shaped, yellowish-brown to dark-brown spots, occurring most frequently upon the face, neck, and backs of the hands. They may appear, however, upon other parts of the body. They are usually discrete, but occasionally may be so numerous as to form close aggregations. Exposure to the sun deepens their color, and they are, therefore, most conspicuous in summer, fading to a great extent or even disappearing in the winter season. Individuals of fair complexion, and especially those having red hair, are most commonly the subjects of these blemishes, although dark-skinned persons are not exempt. Freckles are not congenital, but first appear about the sixth or eighth year, and last usually till after middle age or throughout life. Anatomically the affection consists of a localized increase of the pigment normally found in the skin.

Freckles may be removed by appropriate external applications, but a return to the same condition is almost invariable. Treatment is purely local, and consists in the application of such remedies as will tend to remove or destroy the pigment-containing cells of the epidermis. Corrosive sublimate, in the form of a lotion of the strength of one-half to five grains to the ounce, applied once or twice daily, is one of the most efficient. Another excellent remedy is lactic acid: this is applied to the spots, diluted with one to several parts of water. Tincture of iodine and acetic acid may also be applied with more or less benefit. Ointments may likewise be employed, such as one containing thirty to sixty grains of ammoniated mercury to the ounce. An ointment containing one-half to one drachm each of the subnitrate of bismuth and ammoniated mercury is highly recommended by Neumann. In young children and those with sensitive skins, care should be taken that the preparation employed is not too strong.

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[1] I am indebted to Dr. Milton B. Hartzell, assistant in the Skin Dispensary of the Hospital of the University of Pennsylvania, for material aid in the preparation of the various articles appearing under my name in this work.

NÆVUS PIGMENTOSUS.

Nævus pigmentosus, commonly known as mole, consists of a circumscribed increase in the pigment of the skin, usually associated with hypertrophy of other portions of the integument. When the nævus is smooth and flat, consisting essentially of augmented pigmentation alone, it is designated nævus spilus; if, in addition, it is the seat of an abnormal growth of hair, it is termed nævus pilosus; and when to the excessive pigmentation there is added an increase in the size of the papillæ of the corium, causing the surface to present a furrowed or uneven surface, there results the variety known as nævus verrucosus; if the connective-tissue hypertrophy is excessive, it is designated nævus lipomatodes.

Pigmented nævi vary greatly as to shape and size, being usually, however, circular or oval, and varying in dimension from a pea to a bean, although they may reach or exceed the size of the palm. They may occur upon any portion of the body, singly or in numbers, but are somewhat more frequent upon the back, face, and neck. They vary in color from a light to dark brown or black, and the hair usually found growing upon them may be either colorless, very fine, and short, or deeply pigmented, coarse, and of considerable length. They are usually congenital, but the smooth non-hairy moles may be acquired; the hairy and verrucous varieties are, on the contrary, almost invariably born with the individual. As a rule, nævi are permanent. Microscopical examination shows a marked increase in the pigment in the lowest layers of the rete mucosum, as well as more or less pigmentation in the corium usually following the course of the blood-vessels. In the verrucous variety the papillæ are greatly hypertrophied, in addition to the increased pigmentation.

Treatment, when demanded, consists in removal, either by the knife, by caustics, or by electrolysis. This last is, in the milder varieties at least, perhaps the best method, as it is less likely to be followed by disfiguring cicatrices. In nævus pilosus the removal of the hair by electrolysis is frequently followed by a decided diminution of the pigmentation.

ALBINISMUS.

The term albinismus is employed to designate the condition characterized by the congenital absence, either partial or complete, of the pigment normally present in the skin, hair, and eyes. The individuals in whom this absence of pigment is complete have received the name of albinos. In such the skin is white, the hair very fine, soft, and white or whitish-yellow in color, the irides are colorless or light blue, and the pupils, owing to absence of pigment in the choroid, are red. The absence of pigment in the eyes gives rise to photophobia and nystagmus. Albinos are commonly of feeble constitution and are apt to exhibit imperfect mental development: to this statement, however, there are exceptions.

Partial albinismus is met with most frequently in the colored race, but in exceptional instances occurs also in Caucasians. In this form of the affection the pigment is absent in one or several variously-sized patches. The hair growing in these areas is likewise colorless. The patches, as a rule, undergo no change, but in occasional cases continue to increase in size until a great part of the integument is involved. In rare instances restoration of pigment has been observed.

The functions of the skin are performed in a perfectly normal manner, and microscopical examination shows no departure from normal structure save the complete absence of pigment. Little is known of the causes producing this anomaly beyond the single fact that heredity frequently, but not invariably, plays an important part in its production. The condition is without remedy.

ALOPECIA AREATA.

Definition.—Alopecia areata, also known as alopecia circumscripta, area Celsi, and tinea decalvans, is an affection of the hairy system, in which there occur one or more circumscribed, round or oval patches of complete baldness, unattended by any marked alteration in the skin.

Symptoms and Clinical History.—The scalp is the region most frequently affected. The disease may begin suddenly, without premonitory symptoms, a patch being formed in a few hours; or, as is more usually the case, several days or weeks may elapse before the bald area is sufficiently large to become noticeable. The patches continue to extend peripherally for a variable period, and then remain stationary, or several may gradually coalesce and form a large, irregular area involving the greater portion of the scalp. The skin in the affected regions is smooth, faintly pink or milky white, and at first presents no departure from the normal. Sooner or later, however, the follicles become less prominent, and slight atrophy occurs, the bald plaque being slightly depressed below the level of the surrounding healthy skin. After the lapse of a variable period, the patches cease to extend, the hairs at the margin of the bald areas being firmly fixed in the follicles.

In the beginning of the malady, and for some weeks, the skin of the affected area is perfectly smooth, entirely devoid of hair, presenting an ivory-like appearance; but after a time a fine colorless lanugo, or down, usually shows itself, which may continue to grow until it attains a considerable length and then drops out; or it may remain, become coarser and pigmented, and the patch resume its normal condition. Not infrequently, however, after growing for a time the new hair again falls out, and this may happen several times before the termination of the disease, months or even years elapsing before a definite cure takes place. Occasionally the new growth of hair may be white and remain so; as a rule, however, it finally resumes its normal coloration.

In the large majority of cases the disease is limited to the scalp; but it may invade other portions of the body. The eyebrows and the lashes may be affected, and in rare instances every hair of the whole integument may be involved. Subjective symptoms are rarely present; but occasionally its appearance is preceded by severe headache, itching or burning of the scalp, or other manifestations of disturbed innervation. Neither sex, age, nor hygienic surroundings seem to exert any appreciable influence upon its occurrence. It is, however, probably most common between the ages of ten and forty. Microscopic examination of the skin of the diseased areas shows little or no alteration in its structure.

The etiology is exceedingly obscure. There are two theories as to its causation: one of these regards it as of a parasitic nature and therefore possessing contagious properties, and the other considers it to be of trophoneurotic origin.

Diagnosis.—The only disease for which it may be mistaken is tinea tonsurans, but, with moderate care in the examination of the diseased parts, an error can scarcely occur: the plaques of alopecia areata are smooth, entirely devoid of hair, and free from scales; while those of tinea show numerous broken hairs and stumps, desquamation, and symptoms of inflammation. In doubtful cases recourse should be had to the microscope.

Prognosis and Treatment.—The prognosis in children and young adults is almost invariably favorable, permanent loss of hair being exceedingly uncommon. The uncertain duration, however, must be borne in mind; months and in some instances several years may elapse before complete restoration of the hair takes place. Moreover, the possibility of relapses should not be forgotten.

Treatment should be both local and constitutional. Internally arsenic is perhaps the most valuable remedy, while quinine, cod-liver oil, and ferruginous tonics may in suitable cases often be administered with benefit. Locally such applications as will exert a stimulating effect are to be used. Ointments of tar or sulphur, of varying strength, may be applied for this purpose. The various mercurial ointments are also valuable. The tar oils, either pure or with alcohol, may also be used. Stimulating lotions, containing varying proportions, alone or in combination, of tincture of capsicum, tincture of cantharides, aqua ammoniæ, and oil of turpentine, are also valuable. Frequent blistering of the bald patches is advisable in obstinate cases, and is often of great service in hastening the growth of the hair.

Robinson extols highly the cautious use of an ointment of chrysarobin, twenty to forty grains to the ounce. Galvanization or faradization of the affected parts may be employed, and not infrequently with beneficial effect.

The strength of the application will depend upon circumstances, a mild degree of irritation being desirable. Ointments and oils, if used, should be thoroughly rubbed in, the friction employed being not without value. Watery or alcoholic lotions are usually to be dabbed on, several times over at each application.

For a successful result in alopecia areata, persistent treatment is almost invariably demanded.

DISEASES OF THE NAILS.

The nails may be increased in number, double nails occurring upon a finger or a toe; or they may be augmented in bulk. Rarely they are found in abnormal situations.

As the result of a deviation from the normal direction of growth, the nail may press upon the surrounding tissues, producing varying degrees of inflammation,—*paronychia.*

The matrix of the nail may become inflamed as the result of a preceding eczema or psoriasis, or it may be a manifestation of syphilis. In this affection, known as *onychia*, the tissues about the root of one or more nails become red, swollen, and painful, and suppuration may occur beneath the nails, which are thus loosened and finally cast off. In the non-syphilitic variety resolution may occur before the formation of pus, and the nails be preserved.

Treatment must be directed against the cause. In paronychia the nail should be frequently trimmed and a pledget of lint or cotton be interposed between the edge of the nail and the soft parts adjoining. Astringent powders or lotions may often be employed with advantage.

HYPERTROPHY OF THE NAIL, or *onychauxis*, may be either congenital or acquired. In the latter instance it is usually the result of the extension to the matrix of such cutaneous diseases as psoriasis or eczema, or is produced by constitutional maladies, such as syphilis. The hypertrophy may take place in one or all directions, and this increase may be, and often is, accompanied by changes in shape, color, and direction of growth. One or all of the nails may share in the process.

Treatment consists in the removal of the redundant nail-tissue by means of the knife or scissors, and, when dependent upon eczema or psoriasis, the employment of remedies suitable to these latter diseases. When it is the result of constitutional syphilis, the medication appropriate to this disease should be prescribed.

Atrophy of the nails, or *onychatrophia*, may be either congenital or acquired; most frequently it exists as the result of some local or constitutional disease. The nails are soft, thin and brittle, splitting easily, and are often opaque and lustreless. This condition may result from trauma, or be produced by certain cutaneous diseases, notably eczema and psoriasis, or follow injuries or diseases of the nerves. Syphilis and chronic wasting constitutional diseases may also interfere with the normal production of the nail-substance, producing varying degrees of atrophy. The fungi of the various mycoses of the skin may at times invade these structures and lead to more or less complete disintegration,—*onychomycosis.*

Treatment of atrophy of the nails will depend upon the cause. When it is due to eczema or psoriasis, appropriate constitutional and local remedies should be prescribed. If it is the result of syphilis, mercury and potassium iodide are to be advised. In onychomycosis, an exceedingly obstinate affection, the nails should be kept closely cut and pared, and a one- to five-grain solution of corrosive sublimate applied several times a day. A lotion of hyposulphite of sodium, a drachm to the ounce, is also a valuable and safe application.

NÆVUS, OR BIRTH-MARK.

By LEWIS S. PILCHER, M.D.

Under the term nævus are included all congenital markings of the skin, both those which are visible at birth and those which make their appearance soon after birth. They may be due to simple excess of pigment (macula, nævus spilus, nævus pigmentosus), or to circumscribed hypertrophy of all the dermoid elements (mole, nævus verrucosus, nævus pilosus), or to vascular dilatations of varying degree and extent (port-wine stain, spider-mark, fire-mark, nævus vasculosus, angioma, cavernoma). An extensive birth-mark may unite in itself all these characteristics.

Birth-marks are extremely common: children without blemish of some kind are the exception. In their simplest form of small pigmented spots, or of minute capillary dilatations, they may escape notice altogether. Temporary birth-marks in the form of quite extended bright-red patches may often be noticed, especially upon the forehead, the eyelids, the nose, the scalp, or the nape of the neck of new-born children, which after a few weeks, or months at farthest, undergo spontaneous regression and disappear altogether. The discussion of maculæ and moles calls for but very brief consideration, but the remaining class, vascular dilatations, will present for study a very extensive and important field.

In repeated instances the development and growth of nævi, of all varieties, have been noticed to follow accurately the distribution of certain cutaneous nerves, trigeminus or various spinal nerves, and to be strictly localized on one side of the body: hence the terms nerven-nævus, nævus unius lateris, papilloma neuropathicum. The nævoid degeneration may be diffuse, as in a case reported by Simon of a vascular nævus occupying the territory supplied by the second branch of the left trigeminal nerve, in which the left cheek, the mucous membrane of the left half of the hard and soft palates, and the left tonsil were strongly injected and the coloring stopped exactly at the middle line, or as in cases reported by Neumann, in which even one entire half of the body was darkly pigmented and partly covered with papillary excrescences. Usually, however, the whole field of distribution of the nerve is not occupied, but only multiple islets between which normal skin remains.

Etiology.—The causes of nævi are wholly obscure. Even in the nerven-nævi,—which have been considered by some as plainly the result of a neurosis, in some instances trophic, in others vaso-motor in character,—both the nature of the lesion and its *modus operandi* are entirely hypothetical. No less an observer than Kaposi denies the neuropathic origin even of this class. The fact that vascular nævi frequently occur in the neighborhood of fissures, either temporary fissures, as the branchial clefts, or permanent, as the labial, palpebral, or those for the fingers, caused Virchow to suggest the possibility that slight irritative conditions during embryonal life at the borders of these fissures, where the vessel-development is naturally abundant, might provoke excessive vessel-development, and thus lead possibly to the formation of angiomata, appearing either as congenital conditions, or as growths developing later in life through the awakening into activity of congenital predispositions. This theory, even if accepted in explanation of the production of the nævi occurring in the neighborhood of such fissures, leaves unexplained a very large number of cases which are found in locations distant from fissures. In any case it removes the inquiry only one step backward, and leaves in the dark the primary cause.

Heredity in certain cases may be accepted as the cause of nævi. Prenatal maternal impressions are often claimed as the cause of these marks, and many cases are cited which lend a considerable degree of plausibility to the claim. It is more rational, however, to explain these cases by the principle of coincidence.

Pathology.—The anatomical structure of nævi varies extremely in the different varieties, but in all cases there are to be found changes simply of a hypertrophic character.

Maculæ.—The smooth pigmented spots present abnormal accumulations of pigment in the deeper layers of the rete mucosum, together with more or less great accumulation of pigment in the corium. These flat moles grow only in proportion to the growth of the tissue of which they form a part. The skin is normal in its functions. The spots are simply blemishes in so far as they depart from the natural color of the skin.

Verrucose nævi present in varying degrees hypertrophies of the various elements of the skin, involving in some cases the subcutaneous connective tissue. The growth is always more or less elevated above the surface of the skin, and may vary in extent from a small wart-like growth to widely-extended excrescences covering considerable portions of the body-surface. These more widely-extended hypertrophies, when they involve the subcutaneous connective tissue, are closely allied with elephantiasis. The activity of the elements of the hair-follicles often causes them to be covered with abundant, often coarse hair, and thus to increase their unsightliness and to win for them the appellation of "hairy moles." The hypertrophied sebaceous follicles may produce abundant secretion, whose odor is apt to be disagreeable, a peculiarity which becomes more marked later in life. A striking example of this was reported at the 1888 meeting of the American Medical Association, by Reynolds, of Chicago, in the case of a ten-year-old boy, who had a congenital, elevated, dark-purple verrucose nævus on the left thigh and hip, which nearly encircled the limb and extended from a little below the knee upward as high as the crest of the ilium. The discharge from it was profuse, and of such offensive odor that he was deemed unfit to be in school with other children.

The corneous layer of the epidermis may be greatly thickened, causing the more protuberant portions of the excrescence to be covered with a horny mass, producing the appearance of ichthyosis.

The amount of pigment, always in excess, still presents variations, causing gradations in the color of these nævi from light brown to dark purple.

Vascular nævi present themselves in three broad classes as regards their anatomical conformation, although there is no absolute boundary dividing them, but a gradual transition from one to the other, illustrated by many cases.

1. The more common wine-marks or fire-marks are the result of dilatation of the superficial skin-capillaries. They range in color from a faint pink to bright red and dark purple, according to the extent of the dilatation and whether this has been toward the arterial or the venous termination of the capillaries. Their color disappears momentarily under pressure. They present a smooth surface, with irregular, sharply-defined outlines. The spontaneous regression and disappearance of many of these patches of capillary dilatation which are visible at birth has been already referred to. These temporary patches, however, are, according to my observation, fainter in their color and less strongly defined in their outline than those which are to be permanent. The permanent patches, as a rule, remain unchanged throughout life, growing simply with the growth of the part on which they are situated. They do not extend into the deeper tissues and form small tumors. Nevertheless, as Fox has pointed out, with advancing years there may develop, at points upon their surface, small erectile tumors the size of a pin's head or of a pea, which break the smoothness of their surface.

Weinlechner has reported a case which presents further exception to this rule and illustrates the possibility of peripheral growth. A superficial fire-mark, which was only a speck the size of a linseed at birth, had by the time the child had reached eighteen years of age spread over a large part of the face and neck, and, moreover, had extended to the mucous membrane of the gums, the cheeks, the floor of the mouth, the tongue, the soft palate, and the posterior wall of the pharynx, on the right side.

2. *Simple Vessel-tumors*, Angioma Simplex, Angioma Plexiformis.—The distinguishing characteristic of this group of nævi is that the vessels of which they are composed still possess their own walls. The construction of these tumors is described by Weinlechner as follows: "The simple vessel-tumor is made up of vessels, as a rule, entirely of new formation, seated in the skin or in the subcutaneous connective tissue, which are intricately intertwined, and are dilated and hypertrophied. These are held together by a slight connective-tissue stroma, which in rare instances becomes thickened

into a capsule-like envelope. As a rule, however, dilated vessels are found running out from the tumor into the adjacent tissues, and sometimes isolated, island-like, ectasic spots may be found among these outrunning vessels. The changed appearance of the skin is produced by the dilatation and hypertrophy of its capillaries. The exuberant growth of the vessels is confined often externally to the vessel-districts which belong to existing organs in the skin and the subcutaneous connective tissue, as the hair-follicles, the sebaceous and sweat glands, fat-cells, etc., so that both to the naked eye and under the microscope the tumor has a lobulated formation, each lobe corresponding to the vascular system of one of these organs. The new vessels are produced by outgrowths from the old vessels."[1]

When the capillaries of the corion only are first involved, there is formed a superficial vascular nævus which closely resembles the first class already described, from which the subsequent course alone distinguishes it. They are, however, more commonly minute when first noticed, like a flea-bite, or of the size of a small pea; their borders are not sharply defined; they are prone to extend their borders, in some cases along the surface only, in others inward into the subcutaneous connective tissue, in which case they become converted into distinct vascular tumors. Gross, in his "System of Surgery," relates the case of a child who at birth presented a red spot as large as a dime at the centre of the left cheek. At the end of five weeks it had nearly doubled its dimensions. It was then partially destroyed by an escharotic, after which it took on a more rapid growth, until by the time the child was thirteen months of age it had spread over the whole of the left side of the face, horribly disfiguring the features; it involved the whole thickness of the cheek, and, by the swelling which it had produced of the gum of the upper jaw, was seriously encroaching upon the mouth. A more aggravated case yet is reported by Hulke,[2] in which the nævus, beginning at birth as a few telangiectatic spots in the left groin, gradually spread over the whole surface of the body, acquiring its greatest development on the left half of the body. Repeated bleedings, ulcerations, and erysipelas caused death in the ninth year.

The course of these nævi is more commonly one of slow extension for a while, followed by a period during which they remain stationary; possibly they then shrink away and even may disappear entirely, or on the other hand may be excited into much activity of growth. In illustration of the latter may be cited a case, related by Saint-Germain,[3] of a girl who from birth had had an insignificant violet spot behind her right ear. It remained quiescent until she was ten years of age, when it suddenly took on so rapid a growth that in two years it had become developed into a reddish pulsating tumor, nearly three inches long, two inches broad, and one and a quarter inches high.

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[1] Gerhardt's Handbuch der Kinderkrankheiten.

[2] Med.-Chirurg. Trans., 1877.

[3] Chirurgie des Enfants.

When the primary vascular hypertrophy has its seat in the subcutaneous connective tissue, it forms a roundish, soft, elastic, often apparently fluctuating tumor, which causes the overlying skin to project as an ill-defined swelling from the size of a pea upwards, the skin itself showing no alteration in its color or texture, except possibly that it looks bluish when the tumor is made turgid. With the further extension of the vessel-growth, if the skin becomes involved, its vessels appear dilated more and more, until finally the full appearances of the superficial angioma are superinduced upon the deeper conditions. Thus these growths may begin in the superficial capillaries and later extend into the deeper districts, or the reverse may be the case, so that all degrees of mixed forms may be developed. It should be noted that in some of these subcutaneous tumors there is also an increased formation of fat and connective tissue, making the tumor a mixed angioma and lipoma.

The variation in color of these simple vessel-tumors depends in part on the abundance of the vessels, in part on the thickness of the skin which covers them, and in part upon the relative amount of arterial and venous blood within them. The epidermis is, as a rule, unaltered.

3. *Cavernous vascular tumors*, angioma cavernosa, cavernoma, are vessel-tumors in which the vessel-walls are in part absorbed and the blood circulates in a net-work of spaces. On section they present anatomical conditions identical with those of the corpus cavernosum penis. The cavernous tissue may be more or less distinctly bounded by a sort of capsule of condensed connective tissue, or it may be diffused, losing itself imperceptibly in the adjacent tissues. The stroma of the tumor is formed of the remains of the tissue in which the ectasia has occurred, and the blood-spaces are lined by venous endothelium. They either are connected to large venous trunks, or numerous small arteries and veins sink into their capsules. These tumors usually contain venous blood, but in exceptional cases large arteries feed them. They then pulsate, and give a murmur when auscultated. The favorite site for these growths is the subcutaneous connective tissue, and their size may vary from that of a pea to that of a man's fist.

Cavernous tumors are not infrequently developed after the age of childhood is passed, usually as the result of some trauma, but about one-half of the cases appear in childhood, and most frequently either congenitally or in the course of the growth of simple vessel-tumors. The remarks that have been made as to the relations of the skin in simple vessel-tumors apply equally to cavernous tumors.

Symptomatology and Diagnosis.—The greater number of nævi present no special symptoms other than those superficial appearances which have already been detailed in describing varieties. It is only in the case of purely subcutaneous tumors that any doubt in diagnosis is likely to arise, when in some instances their differentiation from other soft varieties, as cysts, lipomata, sarcomata, may require attention. The pathognomonic diagnostic sign is variation in the size of the tumor. Pressure upon an angioma flattens it and reduces it in size; as soon as the pressure is removed it quickly swells out again to its original size. The act of crying, strong expiratory efforts, a dependent position, cause it to swell still more and become tense. In cavernoma the filling up after removal of pressure is much more gradual than in the case of simple angioma. The degree to which a vascular tumor can be reduced in size by pressure varies, being dependent upon the relative proportion of vessels, or spaces, and stroma. Pain is a frequent symptom presented by cavernoma, and most frequently accompanies the small, more circumscribed forms. Pain induced by the dependent position in such tumors of the lower limbs may make walking impossible.

Location.—The smooth pigmented nævi and the verrucose nævi occur in all parts of the body, and no predilection for certain sites can be observed. Vascular nævi likewise occur on all parts of the body, but the favorite site for them is the head, and especially the face. Thus, of three hundred and thirty-three cases of vascular nævi noted by Weinlechner, two hundred and forty-three were on the head, two hundred of these being on some part of the face, while forty-three were on the scalp. Of the facial nævi, fifty-four were frontal, thirty-five palpebral, thirty labial, thirty-two nasal, twenty-six buccal, fifteen auricular; over the mastoid process were three, on the side of the lower jaw three, on the chin two. There were fifty-six on the trunk, fifteen on the upper extremity, nine on the lower, seven on the neck, three on the genitals.

Prognosis.—Pigmented and verrucose nævi remain unchanged throughout life, as a rule. In occasional instances they become the subject of malignant degeneration late in life. Thus, of fourteen cases of melanoid cancer affecting the skin or subcutaneous tissue, noted by Paget,[1] in ten the disease commenced beneath a congenital pigmentary nævus. In thirty-four cases of melanosis of the skin noted by Pemberton, fifteen commenced in or near a congenital mole. Malignant growths supervening upon vascular nævi are less frequent, but still are of occasional occurrence. The permanence and unchangeableness of the superficial vascular dilatation of the skin, nævus vasculosus, were remarked upon in describing it. The history of tumor-like nævi is, on the contrary, extremely variable. As Holmes has pointed out,[2] very often we see persons in advanced life in whom nævi have remained exactly in the same condition and of the same size as they were soon after birth. Sometimes they wither away and undergo degeneration. At other times, on the contrary, they advance with frightful rapidity, causing horrible disfigurement, or giving rise to hemorrhage which threatens life. Again, after a transient period of activity, they may become stationary, and finally, after standing still for many years, they may again begin to grow. Spontaneous shrivelling or sloughing may be determined by the general debility attending prolonged and exhausting illnesses. The vitality of the nævoid tissue is always less than that of normal tissue, and inflammatory processes, with ulceration or sloughing, are easily provoked in it. Only in the rarer arterial forms of vascular tumor, or in hæmophiles, does the bleeding from erosions of nævi attain much importance.

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[1] Surgical Pathology.

[2] Diseases of Children.

Partial obliteration of a vascular nævus, either by spontaneous atrophic obliteration of its vessels without inflammation, or as the result of inflammation and sloughing with cicatricial contractions, is not uncommon; but the obliteration rarely extends over the whole nævus,—the periphery either persists unchanged or continues to extend. Complete spontaneous disappearance of a nævus even of large size has been reported.[1] It is so rare, however, as to deserve mention simply as a remote possibility.

Nævi are benign growths. The instances in which malignant degenerations have later involved them must be referred to the fact that they present *loci minoris resistentiæ*, favorable to the fixation of the malignant influence whatever it be, just as in many other instances trauma plays the same part in determining the site of a malignant degeneration. After removal, no recurrence takes place, provided all the diseased tissue is taken away. Special mention should be made, however, of the danger to the eye from vascular tumors developing in the deep cellular tissue of the orbit. Not only may the eyeball be pushed out from its place with loss of sight, but sloughing of the cornea may result, necessitating extirpation of the eyeball.

Treatment.—Maculæ may be readily removed by the use of some of the less severe escharotics, as strong nitric acid or ethylate of sodium. Verrucose nævi are best removed by the knife, or, if this is impracticable, by some of the more destructive escharotics, as chloride of zinc or the Vienna paste. Superficial vascular nævi, if they are of small size, are best treated by the actual cautery, the point of a heated needle or of the thermo-cautery being made to penetrate the nævus so as completely to destroy it. Punctate vascular nævi as soon as they are noticed should be at once attacked, for their destruction at this stage is simple and entails no danger, and, though many do not later increase if let alone, yet so considerable a proportion do continue to grow and extend, that the rule should be made general to destroy them all in their beginnings. Extensive superficial nævi, the wine-marks and fire-marks, are not easily removed, except by the use of destructive escharotics which leave cicatrices almost as objectionable as the original nævus. Much improvement in the more unsightly of them may be obtained, however, by careful and persistent treatment. Repeated applications of mild caustics may suffice for the most superficial and light-colored patches. Pure carbolic acid, painted upon the surface, has been praised for this purpose (Fox), its application being repeated weekly until the desired effect has been produced. A solution of corrosive sublimate in collodion, four-per-cent. strength, is a yet more efficient application. The surface of the nævus should be painted with this once daily for four consecutive days, until a thick crust is formed. The healthy skin about it may be protected by a preliminary coat of ordinary collodion. The crust comes away spontaneously after eight or ten days, leaving a granulating surface which heals rapidly under simple dressings, forming a smooth cicatrix that contracts very slightly (Boeing). Ethylate of sodium is a still stronger escharotic. It has been especially praised for the treatment of nævi by B. W. Richardson. In its use the surface of the nævus is first well dried, and is then thoroughly coated with the ethylate, applied with a camel's-hair brush. The application produces some pain, but it is easily borne. A superficial layer of tissue is destroyed by the caustic, which in a few hours forms a thin blackish crust. After the falling of this eschar, repeated applications of the agent may be made until the cure is complete. Nitric acid is valuable as a caustic application in the treatment of small superficial nævi, but it is objectionable in the treatment of the more extended ones, on account of the rough and unsightly cicatrices which follow its use.

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[1] Brodie, Schuh.

Linear scarification is claimed by Balmanno Squire to have accomplished in his hands most satisfactory obliteration, without scar, of very aggravated port-wine marks. He first freezes the portion to be operated on, by means of the ether spray, and then rapidly makes multiple parallel incisions not more than a sixteenth of an inch apart, not more than a sixteenth of an inch in depth, and as long as they can be made quickly and straight. The bleeding is slight, if the cuts are not too deep, and is readily arrested by pressure with blotting-paper. This paper should be gently peeled off before it has dried, and in the direction of the incisions. Special care is to be taken to avoid separating the wound-edges in the scarifications, so that no clot may be found in them. Rapid healing of these incisions, without visible scar, takes place. As soon as the first set of cuts has healed, the process of scarification is to be repeated, and so on again and again until full obliteration of the nævus is secured. At each operation the direction of the parallels should be oblique to that of the parallels of the preceding one. The operation requires great care and skill, and many failures have been reported by other surgeons who have practised it.

Electrolytic tattooing has been successfully used by Fox, and others, in improving, though not absolutely curing, aggravated and unsightly superficial nævi. A single needle, or a number of needles combined in one instrument, is attached to the negative cord of a constant-current battery, from sixteen to twenty cells being required. The needle is inserted into the skin, and the circuit completed by having the patient grasp a moist sponge or electrode attached to the positive cord. The needle should be allowed to remain in the skin from ten to thirty seconds, depending upon the delicacy of the skin and the effect observed. As many punctures of this kind are made as the extent of the nævus requires. At the end of three weeks the ultimate effect will have become manifest. Close inspection will then show the surface treated to be covered with minute whitish dots, which are the cicatrices caused by the destruction of tissue at the numerous points of needle-puncture. The effect is to lighten decidedly the line of the nævoid patch. Repeated applications may be made as the case may require. The treatment is tedious and painful.

Any discussion of the treatment of tumor-like vascular nævi opens up a most extensive field of surgical endeavor, about which a vast literature has gathered. The methods which have been found of value consist of three principal classes, either (1) cutting off the supply of blood to the affected spot, or (2) obliteration of the affected vessels by the excitement of local inflammation, or (3) extirpation of the whole diseased tissue. These methods will be found to be more or less efficient and more or less perilous according to the size and location of the tumor. In the treatment of extensive tumors it is often the case that all these various methods of attack are simultaneously or consecutively resorted to.

Extensive radical procedures which put life in serious jeopardy ought rarely to be resorted to. Nevertheless, in the case of the more serious growths about the face, which cause a disfigurement that must make the future lives of their bearers wretched, the surgeon would be warranted, after having first fairly weighed the possible results of less dangerous methods, in incurring any reasonable risk for accomplishing a radical cure. In still other cases, in which threatening hemorrhage, or pain, or serious disturbance of the function of important parts is produced by the tumor, the surgeon is also justified in taking much greater risks for securing radical cure than if merely cosmetic reasons are to be considered.

With these preliminary observations, I will proceed to the description of each of the three classes of methods of attack, already named.

1. Local Anæmia.—This may be accomplished by compression of the tumor, and to some degree by compression or ligation of the afferent arteries. Tumors lying over bony surfaces, or situated in regions that can be grasped on two sides, as the lips or the point of the nose, may be subjected to compression. The treatment must be prolonged if any good is to be expected from it; but in any event the result is uncertain, while the treatment is tedious and often painful. Ligature of the afferent arteries, either, at a distance, of the main trunk supplying the affected region, or of the branches immediately entering the tumor, has not been found to be a measure from which much permanent benefit is to be expected, and its use should be restricted to cases in which there is immediate danger to life from hemorrhage, and to cases where no other method is available, as in tumors of the orbit. Arterial ligations may, however, be of great service as a preparatory step in operations for extirpation of large vascular nævi. This is their chief *rôle*, which is a very important one. Temporary anæmia of a tumor effected by compression, also, is of value as an aid to agents which are injected into the tissue for the purpose of exciting plastic inflammation.

2. Obliterative Inflammation.—Small, quite superficial nævi may be obliterated by the inflammation following vaccination. Punctures with needles garnished with croton oil or carbolic acid have also been success-

fully used. Rubbing with ten-per-cent. tartar-emetic ointment has had its advocates. None of these agents are of use in the more deeply extending nævi under consideration. There remain, however, four general methods of exciting obliterative inflammation in these tumors,—viz., setons, parenchymatous injections, cautery-puncturing, and electrolysis. Each of these methods deserves more detailed consideration.

Setons.—Holmes, who more than most authors is sanguine as to the progressive atrophy of nævi in the tissues of which inflammation has been set up, gives a correspondingly large place to the use of setons for this purpose. Extensive nævi, the complete removal of which would be dangerous, or undesirable on account of subsequent cicatricial contractions, are the ones in which setons are to be resorted to. Strands of thick silk should compose the seton ; they should be threaded on a needle just large enough to carry them, so that the threads shall themselves fill up the punctures made in their insertion, thus diminishing bleeding. These threads may also be steeped in a solution of perchloride of iron, if the fancy of the operator dictates it. Holmes recommends to pass two or three setons deeply into the tumor, if possible beneath or close to its base. In situations which allow such treatment, it is well to cut the tumor through by two or three strings tied firmly round its whole mass. If the skin is sound, it should be divided previously ; otherwise it should be included in the loop.

Parenchymatous Injections.—Many agents have been used for injection into the substance of vascular tumors for the purpose of producing coagulation and adhesive inflammation in them. To mention them all would be tedious and unprofitable. Reference will here be made only to the agent which is unquestionably superior to all others for this purpose,—viz., perchloride of iron in solution.

The best results with this salt are to be had by the use of a strong solution, 30° Baumé, which is equivalent to about thirty per cent. of the dry salt in the solution. No free hydrochloric acid should be present : should litmus-paper show an acid reaction, it should be neutralized by carbonate of sodium. The injection into the tumor is made with a hypodermic syringe, the needle being thrust well in towards the base of the tumor. The injection may be repeated at one or more points, according to the size of the tumor. Peripheral compression should be made about the tumor during the injection, and should be kept up after it until a firm coagulum has been formed. It is desirable to have the tumor as empty of blood as possible at the time the injection is made, so as to reduce to a minimum the diluting action of the blood and to secure the full irritating effect of the iron on the vessel-walls. The first injection should be made cautiously, not more than two drops being thrown in. Five drops may be used in subsequent injections. Coagulation of the blood in the tumor and plastic inflammation of the vessel-walls and the stroma is the immediate effect of the injection. By the later organization of the thrombus and the contraction of the tissue of inflammatory new formation, the tumor shrinks, and in the most favorable cases is totally obliterated. If, as is not uncommon, after a short time spots that are not obliterated show themselves, they should be at once attacked by renewed injection, and so on, again and again, until definitive obliteration of the whole tumor is obtained; otherwise relapse is possible.

Not infrequently excessive inflammation, with suppuration and more or less extensive sloughing, results. Local necrosis of the skin at the points of injection often occurs. Repeated instances of sudden death during the injection of nævi have been reported. In most of the cases the tumor has been on the face of small children. Most frequently an extension of the coagulum to the right heart was found on autopsy. Embolism of the carotid occurred in one case (West's). From six to ten drops had been injected in all these cases, and in none of them had pressure upon the periphery of the tumor during the injection been made.

Cautery-Punctures.—A red-hot cautery-point may be thrust into the substance of a vascular tumor in various directions, its repetition depending upon the size of the tumor, with the result of exciting obliterative inflammation of that part of it not destroyed outright by the cautery.

Electrolysis.—Consolidation and shrivelling of vascular tumors may be secured by the electrolytic force of the galvanic current. The method as used by Duncan, of Edinburgh, whose reported results are most excellent, is as follows. A current of between forty and eighty milliampères is used; both electrodes, their shanks insulated, are inserted into the tumor. The negative needle, whose destructive force is more powerful and diffuse than that of the positive, is kept in one place merely long enough to bring about a decided effect; it is then moved to another spot, either withdrawing it or not, as the case may require, and so on until the entire mass of the tumor has been subjected to the action of the current. Anæsthesia is desirable. Repetitions of the operation may be made until a final absolute cure is secured. Electrolysis is an efficient substitute for coagulants, while it is less dangerous and more manageable, and, in cases in which it is important to avoid a scar and the time consumed in the treatment is a matter of indifference, it is to be recommended.

3. EXTIRPATION.—This may be accomplished either by the knife, by the ligature, or by escharotics. The thorough extirpation of a nævus, whenever it is practicable without incurring undue risk to life or producing an unsightly cicatrix, is greatly to be preferred to any other method.

The knife is especially applicable for the removal of those vascular tumors which are covered over by unaltered skin, which by being reflected can be saved. The capsule-like envelope which limits many subcutaneous nævi, both simple and cavernous, facilitates greatly their enucleation and removal. Much more difficulty and danger from bleeding attend the removal of those that are diffuse. In attacking a large vascular tumor by the knife, every resource for the control of hemorrhage should be at the command of the surgeon, and in operating about the face the preliminary provisional ligation of the common carotid artery should be done if the tumor is extensive and diffuse. The knife should be made to traverse healthy tissue; larger vessels are to be tied as cut, while capillary bleeding may be checked by the actual cautery or by tampons. Every portion of diseased tissue should be taken away. Primary union should not be sought for, except on the eyelids and about the nose and mouth; but the wound should be left open to granulate, in order that any resprouting of the vascular growth may be easily detected and at once attacked.

The Ligature.—Subcutaneous strangulation of nævi by silk or hempen thread or by an elastic cord is an ancient method of treatment. It has now become largely displaced in practice by other methods, but still has some place as a supplementary procedure. In its application a needle armed with the ligature is passed subcutaneously either around the base of the tumor, if it is small, or so as to embrace consecutive sections of it, if it is large. Then the ends are drawn tightly so as to strangulate the tissues included in the loop. In some cases transfixion of the base of the tumor by hare-lip-pins may be used to prevent the loop from slipping.

The ligature is unnecessary for small tumors; when applied to large tumors it is tedious and painful in its work, and sometimes provokes serious ulcerative and inflammatory symptoms. The écraseur is merely a rapidly-cutting ligature.

Escharotics.—The actual cautery, in the form of either the galvano- or thermo-cautery or the heated iron, is a most valuable and widely-useful agent for the destruction of vascular tumors. The galvano-caustic wire may be made to encircle the base of certain tumors and quickly and bloodlessly to sever their connections. By repeated applications of the cautery in its various shapes, a vascular mass may be safely reduced to a shrivelled eschar, or by the use of the knife-like cautery it may be extirpated without loss of blood. The eschar falls in from six to eight days, leaving a granulating surface that quickly heals under simple dressings.

Chemical caustics may likewise be used to advantage in attacking tumors which by their size or their diffused character, or by reason of the numerous large vessels which feed them, are unmanageable by other methods without too much risk to life. They are of value also for use in destroying recurring growths whenever they may be detected in the surfaces left after attempt at extirpation by other agents. Out of the great number of caustics that have been used, there are but two which are deserving of mention in this connection,—that is, for the extirpation of tumor-like nævi. These are the Vienna paste (caustic lime and potash, equal parts), and Canquoin's paste (chloride of zinc and wheat flour in various proportions). Of these the Vienna paste is the milder and is easily borne, while its action is prompt and easily limited. The sound parts immediately about the part to be attacked should be covered with adhesive plaster, a fenestra in which exposes the nævus. Then the caustic, made into a thick paste with absolute alcohol, is laid on to the thickness of the back of an ordinary table-knife. It is allowed to remain on for from five to thirty minutes, according to the depth of the eschar desired. Then it is washed off with vinegar. The eschar falls in from ten to sixteen days. Chloride of zinc is more energetic, but limits its action strictly to the region to which it is applied. It produces much pain, and morphine is required to be given while it is being used. A concentrated solution of the salt may be made into a paste by mixing it with wheat or other flour, in the proportion of one part of the salt to two, three, or four parts of flour, according to the strength desired. This paste may then be spread upon the surface to be attacked, the borders being protected by adhesive plaster as already directed. The paste is then to be covered over with cotton confined by a strip of adhesive plaster, and the part left untouched until the caustic has exhausted itself. The caustic arrows of Maisonneuve, made by drying the chloride-of-zinc paste and cutting it into pencil-like strips, furnish an excellent means of attacking an extensive, deeply-penetrating tumor. They are used by thrusting them deeply into the substance of the tumor through punctures made previously for them by a sharp bistoury; or through a canula which is first thrust into the tumor, the caustic arrow being then pushed into the canula and held in place while the canula is withdrawn. The number of arrows thus used will be determined by the size of the tumor. The eschar produced by the chloride of zinc is hard, black, and insensible, and comes away in from six to twelve days.

The scar left by these caustics is not very unsightly: still, the destruction of tissue is greater than attends the use of the knife, electrolysis, or the actual cautery, on which account these latter methods are to be preferred whenever practicable. Especially about the face is the use of chemical caustics undesirable.

SYPHILITIC SKIN-AFFECTIONS.

By I. E. ATKINSON, M.D.

The cutaneous lesions of acquired syphilis in children hardly offer peculiarities that justify their special consideration. They correspond closely with those of adult life, presenting, however, in consequence of the delicacy of the skin in infancy and childhood, a marked tendency towards the development of mucous patches in those situations where the ordinary erythematous or papular lesions are exposed to the heat and maceration fostered by peculiarities of infantile apparel and by the apposition of the cutaneous folds formed by redundant subcutaneous cellular tissue. The most favorable situations for the conversion of these lesions into mucous patches are the neck, the axillæ, the groins, the perineum, the buttocks, at the angles of the mouth and nostrils, and at the margins of the anus. In other respects the cutaneous lesions of acquired syphilis in children are of typical character.

When, however, syphilis is inherited, its cutaneous manifestations are such as to offer peculiarities, although these are less striking than the lesions of other organs and parts. The skin usually presents the first signs of inherited syphilis that attract attention. Its lesions may be present at birth; they also constitute important features of late hereditary syphilis. The period of their greatest prevalence is the first three months of extra-uterine life. A syphilitic fœtus aborted as early as the seventh month may already exhibit evidences of cutaneous alterations, in livid and other discolorations and in the softened and macerated condition of the epidermis. Syphilitic children born alive and at term may at birth display the peculiar eruptions of their disorder; more commonly the skin appears to be healthy, an appearance, however, that rarely continues after the first month, the lesions following closely after the peculiar "snuffling" that usually initiates the symptoms, and accompanying the more or less rapidly developing cachexia; for, although at birth these infants often present the aspect of perfect health, with the evolution of their symptoms this cachexia will almost certainly be observed, and will give the child a pitiable senile appearance that almost seems distinctive. The effects are especially marked upon the skin, which becomes drawn and sallow and as if dirty, an appearance that is heightened by the developing eruptions. These eruptions, which coexist to a greater or less extent, may be considered as—

1, Erythematous; 2, Papular (condyloma); 3, Vesicular; 4, Pustular; 5, Bullous; 6, Tuberculous and gummy; 7, Ulcerous.

1. Erythematous Syphiloderm.

According to some writers (Lancereaux, Jullien), this eruption is very uncommon. It is only uncommon if we adopt as the standard the ordinary roseola of acquired syphilis. That of inherited syphilis rarely strictly conforms to this type. It usually begins as discoid or oval, unelevated spots, about the size of the finger-nail, of a pale-red color which fades on pressure. These spots are occasionally livid or violaceous. They first appear upon the lower portion of the abdomen, quickly extending to the extremities and head. They often coalesce to form extensive areas, encircling the neck, for example, like a necklace. These areas may or may not be decked with a thin branny desquamation. They are especially noticeable upon the face, where, as they slowly fade from the cheeks, forehead, and chin, they no longer disappear on pressure, and come to acquire a characteristic muddy or "café-au-lait" appearance. Scattered irregularly and occupying continuous tracts of integument, the pale-red color early yielding to the dull-yellowish or coppery staining, this eruption is extremely common, and constitutes the "macular syphilide" of inherited syphilis. When situated upon parts of the body exposed to irritation, the erythematous patches sometimes become fissured and crusted, or may even be converted into mucous patches; or, after some weeks, they may become infiltrated and gradually transformed into flattened papules.

Upon the palms and soles a diffuse erythematous condition affords a very characteristic feature of the disorder. The integument of the parts is reddened and wrinkled, and covered with a thin desquamation, often in flakes of considerable size. This erythema is very characteristic, and seems to indicate a tendency towards the more severe bullous eruption, or "pemphigus," of these parts.

This erythema or roseola is one of the earliest eruptions. It may be present at birth, when, on account of the bright-red coloration of the general surface, it may elude detection. It rarely occurs later than the third month. Zeissl considers a very late appearance of this eruption an evidence that the child was not infected before birth, but either during or subsequent to delivery. The hyperæmia is often very evanescent; the characteristic pigmentation succeeding it may persist for a long time. A non-specific erythema is often seen upon the nates, groins, and other parts of syphilitic children, induced by the same exciting influences that may evoke it in non-syphilitic children.

2. The Papular Syphiloderm.

This is also an early manifestation of inherited syphilis. It may be present at birth, alone or associated with other symptoms, or it may, and usually does, develop during the first weeks of extra-uterine life; or it may appear first at a somewhat later period than the erythematous syphiloderm. It is usually scattered over a wide area as the lenticular papule (the grouped miliary papules are much less common). They are discrete, and rarely exceed the size of a hemp-seed. Occasionally only a few scattered lesions can be discovered. Their color is often a pale red; generally, however, they rapidly acquire the brownish-red or coppery hue characteristic of syphilitic eruptions. The papules are distributed over the trunk and extremities with more or less symmetry, and, like the eruptions in acquired syphilis, hardly ever excite any sensation of itching. After a while they often become surmounted by thin scales. This is especially the case upon the palms and soles, where they are broader and flatter, and where they may coalesce into irregular patches with desquamation of thin bran-like flakes. Where folds of the skin are in contact or when the lesions are kept moist by wet napkins, etc., the papules exfoliate their more superficial layers of epidermis, and frequently become converted into mucous patches, the differences of the lesions being due to location and environment, and not to any essential dissimilarity. Such patches may be observed about the mouth and nares, the folds of the neck and groins, but more especially in the region of the nates, the anus, the perineum, and the external genital organs. About the mouth and nares these lesions often become deeply fissured, involving the destruction of connective tissue. In after-years the linear cicatrices remaining after the healing of these become tell-tale witnesses of the inherited vice. In the absence of treatment and of cleanly attention, these mucous patches may undergo great development, coalesce, and form elevated surfaces (condylomata lata) of smooth grayish aspect, emitting the characteristic malodorous secretion. Under treatment the syphilitic papule and condyloma disappear with more or less rapidity, leaving a copper-colored pigmentation that may persist for months. In infants more than six months old, the papular syphiloderms, when of the pale rose-red variety, may closely simulate papular eczema, with which eruption, indeed, it may be associated. The lesions are usually more discrete, evoke but slight pruritus, and are associated with other characteristic symptoms.

3. The Vesicular Syphiloderm.

4. The Pustular Syphiloderm.

These eruptions, being degrees of the same pathological process, may be considered together. The vesicular syphiloderm, except as a stage of the more common bullous eruption, is one of the rarest cutaneous lesions of inherited syphilis. It occurs early, is not widely distributed, and is usually associated with other eruptions. The vesicles develop upon the palms and soles (where they are isolated, and rapidly become opaque or purulent), the back and abdomen, the forearms, the thighs, following a similar course, and upon the face, where they are most often encountered on the cheeks and about the angles of the mouth. Here they are conical, not larger than pinheads, and situated upon a hyperæmic, infiltrated base of dark-red or coppery color, and rather closely aggregated. They do not extend rapidly, but after some days rupture or are ruptured, and form surfaces of inflammatory infiltration, covered with thin straw-colored or brownish crusts, beneath which some superficial ulceration may take place. Or they may gradually become pustular. This suppuration may be regarded as an accident in the course of the syphilitic lesion, arising from the engrafting of the pus-organism. The pustular syphiloderm may occur primarily, and also forms one of the early symptoms of the disease. It occupies by preference the same parts favored by the vesicular eruption, the palms and soles, the forearms and thighs, the back and buttocks, the face and head. It may spring from pre-existing papules or vesicles. The earlier the appearance of this lesion the graver will be, usually, the course of the malady. Upon the palms and soles and trunk the eruption tends to assume a dome shape. It is situated upon dull-red or coppery areolæ, and is discrete. Upon the face the pustules are generally acuminated and aggregated upon infiltrated areas. By extension they may coalesce, or remaining discrete they may rupture and form thickish scabs, which may cover the parts invaded with an unsightly crust, under which ulceration of greater or less degree may take place. Upon the palms and soles the rupture of the pustules may be followed by excoriations and sometimes by deep fissuring. A more formidable termination of the vesicular and pustular syphiloderms is their transmutation into the bullous syphiloderm.

5. The Bullous Syphiloderm.

This grave eruption, which is often present at birth, has been called, injudiciously, "syphilitic pemphigus." It is not at all uncommon. In one hundred and ten syphilitic infants Casati noted "pemphigus" ten times. It may form as early as the sixth or seventh month of intra-uterine life, and only exceptionally develops later than the tenth or twelfth day after delivery. Its situations of predilection are the palms and soles, extending often to the legs and forearms, but not frequently invading the head and trunk. It is symmetrical. It marks an extreme degree of syphilitic infection, and is of very unfavorable augury. The blebs develop irregularly. They begin as small areas of dusky or violaceous infiltration, which after a day or two become surmounted by one or more small vesicles, or, rarely, pustules. These gradually enlarge, to form, after a few days, pea-sized to bean- or even pigeon's-egg-sized blebs. These blebs sometimes have irregular margins from coalescence, and are seated upon dull-red surfaces. At first serous, their contents become turbid and finally purulent or even sanguinolent. By degrees their walls become flaccid and collapse, or are ruptured, and form more or less extensive crusts of dirty-brownish or even blackish color. More frequently these are stripped off by friction or by the discharge, and expose livid secreting surfaces, which may ulcerate or become covered with pseudo-membrane or gangrenous. In favorable cases they re-form a healthy epidermis. The evolution of these lesions accompanies a rapidly-increasing cachexia, and death not unfrequently supervenes after a few days. When treatment is followed by favorable results, new lesions cease to develop and old ones gradually heal, leaving behind a coppery or violaceous, very persistent pigmentation. In severe cases, blebs may form upon the general surface. A bullous eruption, however, which first appears after the second week after birth will probably prove to be not syphilitic. After the twelfth week the bullous syphiloderm is rarely encountered, relapses occurring with great infrequency. In non-syphilitic bullous eruptions in infants the palms and soles are not often invaded, but rather the trunk and limbs. The blebs are larger, their liquid contents clearer, their bases less deeply colored; desiccation is more rapid, and, for a long time at least, there is a notable absence of cachexia. Some exceptions are observed, however. Labtat has recorded a general syphilitic bullous eruption present at birth, not involving the palms and soles, where the epidermis was fine, smooth, shining, but free from lesions.

The cutaneous lesions thus far enumerated are those characteristic of the earliest period of extra-uterine inherited syphilis, and are often simultaneously present. Of these, the erythematous or macular and the papular eruptions are the most common; the vesicular eruption is rare and limited in amount. Concurrently with the eruptions the child generally shows unmistakable evidences of the progress of the disorder, in the development of those other symptoms which appertain to the inherited disease; or, yielding to the influence of treatment, the cutaneous symptoms gradually disappear as the general health improves. They may never redevelop. More commonly, however, relapses occur. During the first two years these consist principally of erythematous or papular eruptions. In cases of weak infection the early symptoms may be so insignificant as to attract but little attention, and a period of quiescence ensue which may extend into or beyond the second dentition; or the frequently-recurring manifestations may begin to exhibit features that are comparable to those of tertiary syphilis. As these come into prominence, the earlier cutaneous symptoms rarely or never occur.

6. The Tuberculous Syphiloderm.

7. The Gummy Syphiloderm.

8. The Ulcerous Syphiloderm.

Taylor has recorded the tuberculous syphiloderm as having occurred as early as the sixth month. This is exceedingly rare. It is not often observed before the end of the first dentition. More commonly it is deferred until the period of second dentition or later, when it becomes a prominent symptom of "syphilis hereditaria tarda." Fournier noted in two hundred and twelve cases of late hereditary syphilis, cutaneous manifestations fifty-six times. These developed from the fourth to the twenty-eighth year, most frequently between the tenth and nineteenth years. The lesions now under consideration usually appear when the stamp of hereditary syphilis has been impressed upon the body of the patient with distinctness. At times, however, they may afford almost the only signs of the disease. The lesions differ somewhat in their course from those of acquired syphilis. Their seats of predilection are the face, particularly the cheeks and nose, and the anterior surface of the legs (Fournier). They are hardly ever widely disseminated, and tend to form close aggregations of crescentic, semilunar, or horseshoe shapes, rarely circular. They appear as tubercles or small gummy tumors, varying from hemp-seed to chestnut size, and are almost painless. The gummy tumors form in the subcutaneous connective tissue, and involve the true skin secondarily. The lesions slowly acquire the peculiar violaceous or coppery color, and remain dry and scaly, or, what is more common, ulcerate. When subcutaneous, the nodules, as they approach the surface, begin to fluctuate, become livid, and finally break down, forming an ulcerating sore with free discharge, excavated margins, and yellowish pultaceous surface. Some authors (R. W. Taylor, Hyde) describe a furuncular eruption in inherited syphilis. A close consideration of the described characteristics hardly justifies acceptance of this form, the resemblance to furuncle being only apparent and not disguising the true relationship to the tubercular or gummatous process.

The process of ulceration in these lesions, however, is often much slower than in acquired syphilis. When tubercular lesions ulcerate, by the coalescence of the group, a crescent or segment of a circle is formed by the ulcer. This may remain quite superficial and comparatively inactive. The coloration of the original lesion may have been pale and but faintly suggestive of the syphilitic process. These circumstances often suggest to the observer lupus rather than syphilis. The term "syphilitic lupus" is an unfortunate but suggestive title for this disorder. Ulceration thus beginning becomes steadily though slowly progressive, and, persisting for months, or even years, may destroy large surfaces, may indeed entirely efface the features, all the while more closely simulating lupus than syphilis except in amenability to specific treatment. This form of ulceration is particularly observed upon the face. Roussel has included among the localities especially affected by the tertiary syphilis of young people, the temporal region. He considers the temporal ulcer a peculiarly diagnostic sign. This ulcer affects the vicinity of the angle of the eye, and is often falsely attributed to scrofula. The ulcerations often seen about the navel, armpits, and groins in infants demand great circumspection in diagnosis. So also do those sometimes occurring upon the heel near the insertion of the tendo Achillis and preceded by desquamation and gangrenous suppuration. These are not necessarily due to syphilis, and often arise from other causes (Casati).

Ulceration following the suppuration of subcutaneous gummy nodules most often occurs upon the anterior surface of the leg, and does not notably differ from similar ulceration in acquired syphilis.

Syphilis hæmorrhagica neonatorum, which has recently attracted much attention, more particularly through the labors of Mräcek, does not offer any cutaneous symptoms differing from those of ordinary cutaneous or purpuric hemorrhage.

Nails.—As the hands and feet so often become the seat of the eruptions of hereditary syphilis, so the nails not unfrequently participate in these processes. The lesions may be either moist or dry. The moist eruptions are usually pustular, and invade at first the nail-fold. They may disappear without injury to the nail. Suppuration may, however, involve the matrix. During its progress the nail becomes raised from its bed and is thrown off. The terminal phalanx of the finger becomes club-shaped. The texture of the regenerated nail is spongy and brittle. Several successive nails may thus be exfoliated, and when the inflammatory action finally subsides the nail may remain permanently misshapen and abnormal. Several fingers and toes may be affected. The dry form of this syphilitic onychia seems to be atrophic. Van Harlingen has described an interesting form of this atrophy in an infant three weeks old. In onychia associated with desquamative erythema of the hands, the nail-fold is reddened and swollen, the nail becoming streaked and fissured.

The Hair.—The loss of hair that follows the pustular and ulcerative lesions of the scalp and depending upon destruction of the hair-follicles is permanent. Temporary alopecia is not unfrequently observed in congenital syphilis of infants. It may be general or circumscribed, and is similar to ordinary syphilitic alopecia of the acquired disease. It is not permanent.

Diagnosis.—The erythematous syphiloderm may be recognized by its general and symmetrical distribution, its persistent course, and its early assuming a ham-, or coppery, or "café-au-lait" coloration. The simple erythematous eruptions of infancy are of a rose-red color, are often elevated, and are very fugitive. They rarely desquamate. An erythema intertrigo is very commonly observed in the course of infantile syphilis. It is excited by the irritation of specific lesions, by filth, by the maceration of discharges. It is more diffuse, and attacks especially the creases of the skin, as those of the neck, the groins, the thighs, etc. The papular syphiloderm is general, its lesions are discrete, are usually not situated upon a hyperæmic base; they are without pruritus, and assume early the specific coloration. These conditions serve to distinguish it from papular eczema, the only affection with which it is likely to be confounded. The vesicular and pustular eruptions are rarely extensive, and may be diagnosticated by their specifically pigmented bases. The bullous syphiloderm is frequently mistaken for pemphigus. The syphilitic eruption occurs nearly always at birth or during the first fortnight afterwards. It affects by predilection the palms and soles. Its lesions are irregular, often flaccid, speedily become purulent, and after rupture not uncommonly ulcerate. They are accompanied by an early cachexia. They are rarely seen after the twelfth week, and are almost never observed in relapses. Simple pemphigus does not occur during the early months of infancy. The blebs invade the trunk and members freely; they are tense and not surrounded by pigmented or infiltrated areolæ, and are not, at least for a considerable time, accompanied by cachexia. The tuberculous syphiloderm is especially apt to be mistaken for lupus. It is, however, usually more speedily and deeply destructive than this affection. The two eruptions especially attack the same parts, and when the syphilitic lesion progresses slowly and superficially the diagnosis may at times only be cleared up after the effects of specific therapeusis have been observed. It should be remembered that lupus attacks deep tissues with much less energy than syphilis. Gummy tumors and ulcerations in hereditary syphilis present, for the most part, the same characteristics as in the acquired disease.

The difficulties of diagnosis of the cutaneous manifestations of hereditary syphilis, which of themselves might often prove formidable, are greatly diminished by consideration of the concomitant symptoms presented by the specific lesions of other parts and organs, which are discussed in the article devoted to the general description of hereditary syphilis.

PARASITIC DISEASES.

BY HENRY W. STELWAGON, M.D.

TINEA FAVOSA.

Definition.—Tinea favosa, or favus, is a contagious disease of the skin, due to the presence in the cutaneous structures of the vegetable parasite the achorion Schönleinii. Its usual seat is the scalp, although any part of the integument may be attacked. It is characterized by variously-sized, circular, concave, yellow crusts, which are usually pierced by hairs.

Symptoms and Clinical History.—Favus begins with the formation of small circular hyperæmic or slightly inflammatory patches, attended by moderate itching and epidermal desquamation. After a short time minute, slightly-elevated, yellowish points appear, which, increasing gradually in size, become cup-shaped, and are, as a rule, pierced with one or several hairs. In the early stages of their formation the individual crusts are covered by a thin layer of epidermis. At the end of ten or twelve days the crusts present a characteristic appearance: they are round, sulphur-yellow, depressed in the centre, and not infrequently show concentric striation. As these cups, or "favi" as they are sometimes called, continue to grow, they tend to coalesce, and may in this manner eventually cover considerable areas of the scalp. They are somewhat firmly attached, and, on removal, the skin beneath is found to be slightly depressed, smooth, and shining, and with, in cases in which the disease has lasted some time, evidences of atrophy. Varying degrees of inflammation may be present, and occasionally pus may be found beneath the crusts. Atrophy and even scarring may, in severe and long-continued cases, result, in consequence of the pressure from the masses of fungus. The crusts usually have a peculiar musty odor. The hairs in the affected areas also soon undergo change: they become dry, brittle, and break off, or fall out. In old cases patches of baldness are sometimes seen, the follicles having been destroyed either through atrophy or inflammation. In some instances the cervical lymphatic glands are swollen, and may even suppurate. Abscess-formation in the scalp may also occasionally occur as a complication. Upon the general integument the appearances of the disease are about the same as upon the scalp. Itching is usually present to a moderate degree, but in exceptional instances this may be a prominent symptom.

In rare cases the parasite invades the nails, and these structures lose their lustre, and become thickened and brittle, their free margins breaking readily. The nails are, however, rarely invaded primarily, the fungus finding a lodgement here through scratching other parts affected.

Etiology.—Favus arises from the presence in certain parts of the integument of a vegetable parasite named by Remak, in honor of its discoverer, the achorion Schönleinii. It is contagious to a marked degree, but susceptibility varies, a certain predisposition or unknown condition of the skin seeming necessary for its development. Children are more especially liable to contract it. The lower animals are not infrequently subjects of the disease, and it is probable that in many instances it is contracted from this source. It is, moreover, essentially a disease of the poor and ill-cared-for, examples of it among the better classes being exceedingly rare.

Tinea favosa is a dermatomycosis having its seat in the hair-follicles, the hair, and the epidermis, more especially in the superficial portion immediately beneath the corneous layer. The fungus belongs to the order of moulds, and is composed of mycelium and spores, of which the crusts are almost entirely made up. The mycelium consists of curved or straight, in some instances branched, tubes having either a homogeneous structure or containing spores, the varying appearances depending upon the stage of development of the individual elements. The spores are minute, round or oval, shining bodies, and are distributed throughout the meshes of the mycelium. Both the mycelial threads and spores are usually present in great profusion. The hair, as well as the follicles and upper layers of the epidermis, are penetrated by the fungus. (See Fig. 1.)

Diagnosis.—The diagnosis of favus offers ordinarily no difficulty. The yellow color of the crusts, their circular cup-like shape, their friability, and their peculiar musty odor, are usually characteristic. In old cases, and especially in those attended with pus-formation, it may be confounded with eczema, but the peculiar crusting, the involvement of the hairs, and the presence of more or less baldness, often with atrophy and superficial scarring, will serve to distinguish it from this affection. It resembles tinea tonsurans only in its involvement of the hair and the consequent alopecia, but in ringworm there is scaling instead of crusting, nor is there the atrophy or scarring of favus. In doubtful cases a microscopic examination will serve to differentiate. For this purpose a small piece of the yellow crust is placed upon a slide, moistened with liquor potassæ, and examined with a power of from three to five hundred diameters.

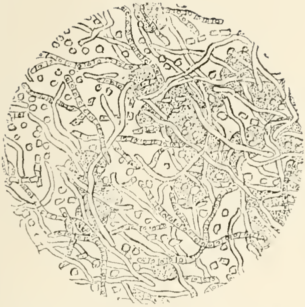
Prognosis.—Favus is a curable disease, but the length of time required to effect a result depends upon the extent of surface involved, and more especially upon the duration of the disease. Upon the scalp, a cure in four to ten months, in an average case, may be considered a good result. Recent cases respond much more quickly than those in which the disease has been long continued. In these latter instances there may be more or less permanent baldness. Upon non-hairy parts of the integument favus is usually readily and quickly cured; when affecting the nails, however, it proves obstinate.

Treatment.—The treatment of favus of the scalp must be energetically carried out if a result is to be expected. The crusts are to be removed by means of oil applications and soap-and-hot-water washing. In cases in which the crusts are more or less tenacious, instead of ordinary soap, sapo viridis may be employed with advantage. Subsequently the scalp is to be washed only at intervals of several days, in order that the remedy used may thoroughly soak into the diseased parts. After removal of the crust, depilation and parasiticides are to be employed. Depilation may be practised in two ways. In those cases in which a great part of the scalp is involved, drawing the hair between the thumb and the side of a comb is advisable, the diseased hairs usually coming away with slight traction. If the area of disease is limited, however, the hairs are best extracted by means of the forceps or tweezers. This latter is, of course, a much more thorough method than the former, but it is also more tedious. This should be practised each day, and a parasiticide applied immediately afterwards. In all cases, however, the remedy should be well applied at least twice daily. The most valuable remedies are corrosive sublimate, in the strength of one to four grains to the ounce of alcohol-and-water; oleate of mercury, in ten- to twenty-per-cent. ointment; sulphur ointment; citrine ointment with one to three parts of lard; and carbolic acid, one to three drachms to the ounce of lard or glycerin. Tar ointment is also valuable. In conjunction with active treatment of the diseased areas, a saturated solution of boric acid, or a strong carbolic-acid lotion, two to four drachms to the pint of water, is to be employed for application to the whole scalp for the purpose of preventing the spread of the disease. At the end of four to six weeks treatment should be intermitted for several days, in order that the effect of the remedial applications may be ascertained. For favus upon the general surface the same remedies, somewhat weakened, are employed, and usually with a prompt result. In favus of the nails, the oleate of mercury and corrosive-sublimate solutions seem to be the most efficacious. These parts should, moreover, be kept thoroughly cut and scraped.

TINEA TRICHOPHYTINA.

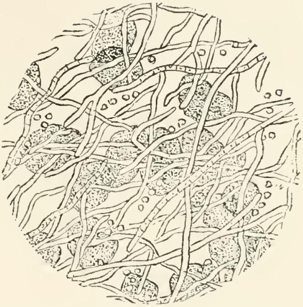
Definition.—Tinea trichophytina, or ringworm, is a contagious disease of the skin, due to the presence of a vegetable parasite, the trichophyton. It varies considerably in its clinical aspects according to its seat, and in consequence of these differences the three varieties, tinea circinata (tinea trichophytina corporis), tinea tonsurans (tinea trichophytina capitis), and tinea sycosis (tinea trichophytina barbæ), demand, for practical purposes, separate description. The last-named variety, being obviously confined to adults, will not be considered here.

FIG. 1.



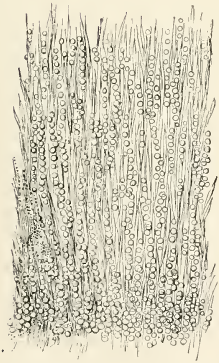
ACHORION SCHÖNLEINII, THE FUNGUS OF FAVUS. × about 450.

FIG. 2.



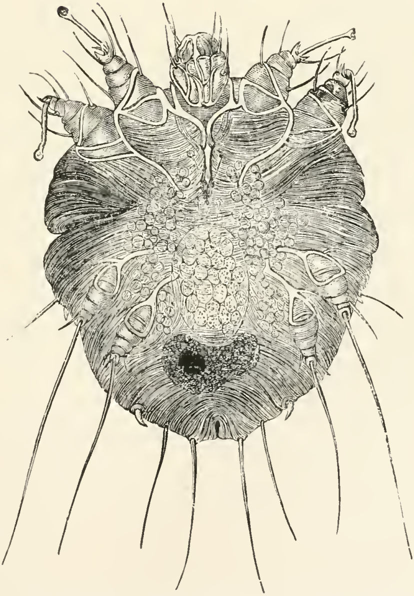
TRICHOPHYTON, THE FUNGUS OF RINGWORM, AS FOUND IN THE EPIDERMIC SCRAPINGS. × about 400.

FIG. 3.



TRICHOPHYTON, THE FUNGUS OF RINGWORM, AS FOUND IN A BROKEN-OFF HAIR-STUMP, the hair being thoroughly disintegrated by the invasion. Spores and chains of spores. × about 400.

FIG. 4.



ACARUS SCABIEI, FEMALE, FECUNDATED: VENTRAL SURFACE. × 300. A matured ovum is visible within the body. (After Kaposi.)

The fungus consists of mycelium and spores. The former occurs as straight or curved tubes, sometimes branching, which as they develop become transversely divided by septa, and within these the spores are formed and after a time set free. The spores are small, shining, round or oval bodies, which in the process of development first show a projection of the cell-wall, and the projection increases until the round spore has become a short tube. (See Figs. 2 and 3.)

TINEA CIRCINATA (TINEA TRICHOPHYTINA CORPORIS).

Symptoms and Clinical History.—Tinea circinata, or ringworm of the body, begins as a small, hyperæmic, scaly patch, circular in shape, sharply circumscribed, and slightly elevated above the level of the surrounding skin. Occasionally the inflammatory action is sufficiently great to lead to the formation of small papular or papulo-vesicular elevations, usually noticeable about the margin. As the patch extends centrifugally the centre becomes less scaly and hyperæmic, and in consequence the resulting lesion is ring-shaped. When fully developed, a patch appears as a round, slightly scaly, somewhat hyperæmic ring, with a more or less clear centre. As clinically observed, this ring-formation is, except in rare instances, invariable. As usually met with, they vary in size from a dime to a silver dollar. Several contiguous rings may coalesce, forming gyrate or crescentic scaly patches. There may be one or more patches present in the single case. As observed in the United States, the number of patches is, as a rule, small, usually three to ten; in some other countries, however, the whole surface is at times invaded. The parts which are naturally more exposed to contagion, as the face, neck, hands, and forearms, are most commonly attacked, but no region is exempt. Itching is usually present in ringworm, but it is rarely a prominent symptom. The disease may run an acute course, disappearing spontaneously in a few weeks, but much more commonly it continues, if untreated, an indefinite period. Ringworm of the body may be associated with ringworm of the scalp. The nails also may be the seat of the disease (tinea unguium), usually contracted from scratching other affected parts. They lose their lustre, become dry and brittle, and show a marked tendency to split longitudinally.

Etiology.—Tinea circinata is caused by the growth of the fungus in the corneous layers of the epidermis. It is highly contagious, being readily communicable from person to person by direct contact or through the medium of various articles of the clothing or of the toilet. The degree of susceptibility, however, varies considerably. It is probably not infrequently acquired from the lower animals, horses, dogs, and cats being also subject to the disease. It is confined to no age, but is by far most common in children. Sex is without influence.

Diagnosis.—While the diagnosis is usually quite easy, yet there are certain diseases, more especially eczema, psoriasis, and seborrhœa, which may more or less closely resemble it. From eczema it is to be distinguished by its circular shape, the sharply-defined margin, the peripheral extension, and the slight degree of inflammation. The circinate patches of psoriasis bear some resemblance, but the marked scaliness and the inflammatory symptoms, together with the presence of ordinary psoriasis-spots, will serve to differentiate. Seborrhœa upon the trunk sometimes presents patches in some respects similar to ringworm, but the greasy character of the scales, and the evident involvement of the sebaceous glands, are usually characteristic of that disease. In all cases of doubt, however, recourse should be had to the microscope. For this purpose the scales are taken from the margin of the patch and moistened with liquor potassæ, and after a few minutes are examined with a power of three to five hundred diameters. An examination of several scrapings should be made before concluding that no fungus is to be found, as in this variety of ringworm the parasite is apt to be scanty. (See Fig. 2.)

Treatment.—The treatment of tinea circinata is usually attended with a rapid result; it is only in exceptional cases that the disease is obstinate, more especially in strumous and debilitated subjects. The remedy should be applied at least twice daily. If an ointment is employed, it should be thoroughly rubbed in; if a lotion is used, it should be dabbed on the patches for several minutes at each application. Hyposulphite of sodium, in solution or ointment, a drachm to the ounce; corrosive sublimate, one-half to four grains to the ounce; sulphur ointment, full strength or weakened with one or two parts of lard; ammoniated mercury ointment, full strength or weakened, may be mentioned as among the most useful applications. For obstinate cases, painting the patches with collodion containing a drachm of chrysarobin to the ounce, or with the tincture of iodine, or applying a chrysarobin rubber plaster, will prove effective. In these cases, also, a solution of corrosive sublimate in tincture of myrrh or benzoin may be employed in the same manner. These stronger remedies, however, should always be applied with great care in infants and young children, and should never, in fact, be resorted to unless the milder measures have been used without result. In strumous patients, if the disease prove obstinate, a favorable result from local treatment may be influenced by the administration of appropriate internal remedies, such as cod-liver oil, iron, and other alterative tonics.

The nails when affected should be kept closely cut and scraped, and one of the above ointments or lotions frequently applied. Attacking these parts, the disease is, as a rule, rebellious, and demands energetic treatment.

TINEA TONSURANS (TINEA TRICHOPHYTINA CAPITIS).

Symptoms and Clinical History.—Tinea tonsurans, or ringworm of the scalp, presents itself as rounded scaly patches of variable size, in which many of the hairs have fallen out of the follicles, while others are broken off close to the skin, producing areas of more or less complete baldness studded with short stumps of hairs. It begins, like ringworm of the general surface, as small, circular, hyperæmic, slightly-scaly patches, usually somewhat elevated, and, as a rule, sharply defined. Occasionally ill-developed papules and vesicles may be found at the margin of the patches. There may be one or more areas, several, as a rule, being present in a single case. As long as the patches remain discrete, which obtains in most cases, they preserve their circular form, but not uncommonly as they extend peripherally they unite, forming irregularly-shaped areas which may cover the greater part of the scalp. Patches of the disease may also be present upon other parts of the body. Slight itching is usually present, but this is rarely so severe as to be a prominent symptom.

Signs of inflammatory action, which may be noticeable in the beginning, are rarely present when the case comes under the notice of the physician. At this time the disease appears as a grayish-white, rounded, circumscribed patch, more or less bald, with scattered hair-stumps. The hairs undergo alterations early in the disease. They become dry, are without their usual lustre, and are exceedingly brittle, falling out or breaking off with an irregular brush-like fracture. In most cases, upon close examination of the involved areas, minute whitish or grayish, scaly elevations, composed of epidermic débris and fungus-elements, are seen projecting from the mouths of many of the follicles, producing an appearance resembling goose-flesh.

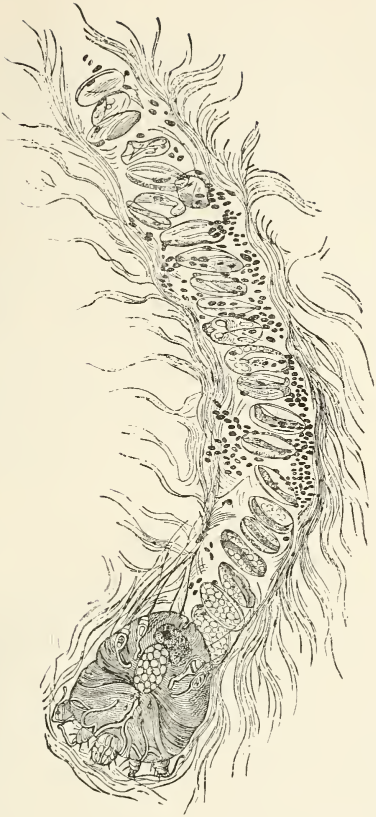
In some cases the disease may exist in the form of small disseminated patches, each patch involving a few or a limited number of follicles. In this form, as the scaliness is slight and the number of stumps small, the disease may readily escape detection unless great care is exercised in the examination of the parts. Occasionally, more especially in strumous subjects or in those whose powers of resistance have been weakened, the inflammatory action may be severe, the affected parts becoming red, elevated, swollen, puffed, and painful; the hairs falling out, and from the distended follicles a sticky mucoid or muco-purulent fluid escaping,—tinea kerion. The severe inflammation acts, in some cases, destructively to the fungus, and a spontaneous cure results.

Tinea tonsurans is rare after puberty: it is essentially a disease of childhood, being almost, if not entirely, unknown in the adult. It is met with in all classes of society, but is obviously more common among the poor and neglected. It occurs frequently in schools and other institutions, and is communicated either by direct contact or, what is probably more common, by means of caps, hair-brushes, etc. The fungus is to be found growing in the epidermis, in the hair-follicles, and in the shaft of the hair. In this last named may be found a profusion of spores, but few, if any, threads. The hair undergoes disintegration, the growth of the parasite forcing its elements apart and rendering it lustreless and brittle.

Diagnosis.—The diagnosis may be usually made without difficulty. The rounded, marginate, scaly plaques, from which many of the hairs have fallen, the numerous broken-off hair-stumps, the peculiar appearance of the affected part produced by the minute projecting cones of epidermic scales, are features sufficiently characteristic to prevent error. Alopecia areata resembles it only in the loss of hair and usually the shape of the patches, but beyond these symptoms the two diseases have nothing in common. In doubtful cases a microscopic examination of the stumps or scales will be decisive. For this purpose the scrapings or hair-stumps should be placed upon a slide, moistened with liquor potassæ, and then examined with a power of three to five hundred diameters. (See Figs. 2 and 3.)

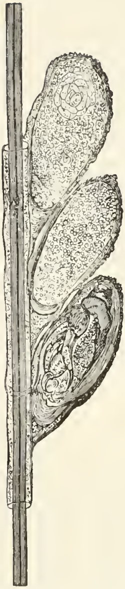
Treatment.—The prognosis as to the ultimate cure of the affection is favorable, but it is not infrequently exceedingly rebellious to treatment. Especially in schools and children's asylums, in which patients are usually pale and weakly, is it apt to be obstinate. Unless thoroughly and perseveringly treated, repeated relapses will occur. In exceptional cases the disease, for a time, seemingly defies all remedies, increasing and spreading under the most energetic treatment. In an average case a cure may usually be effected in two to six months. It is advisable, conjointly with active management of the diseased areas, to make frequent application of an efficient parasiticide to the whole scalp, in order that the spread of the disease may be prevented. For this purpose, a saturated solution of boric acid, a two-per-cent. solution of carbolic acid, or a weak lotion of corrosive sublimate may be employed. The scalp should be washed only at intervals of several days, in order that the remedies used may thoroughly permeate or soak into the parts. Cutting the hair closely, while not absolutely necessary, greatly facilitates treatment and is always to be advised. Depilation of the affected parts should be practised, and, though troublesome, is of great value in expediting the cure, as by their extraction the fungus within the hairs is removed, and the remedy has easier access to the follicles and is thus brought into contact with the deep-lying fungus. The ointment or lotion chosen should be applied at least twice daily. The number of so-called parasiticides from which a selection may be made is a large one, but success depends, in a great measure, not so much upon the special parasiticide chosen as upon the thoroughness and perseverance in its application. At the same time it must be stated that remedies often act differently in different cases, and a change from one to the other may at times be made with advantage. In cases in which the disease is more or less limited, and the attendants intelligent, the most useful remedy is a lotion of corrosive sublimate, two to five grains to the ounce. Carbolic acid, one or two drachms to the ounce of glycerin or ointment, is also often satisfactory. Oleate of mercury, in the form of an ointment, ten to twenty-five per cent. strength, may often be employed with good effect. Sulphur, citrine, tar, and ammoniated-mercury ointments, either alone, or several combined, deserve favorable mention. Chrysarobin, a drachm to the ounce of collodion or gutta-percha solution, or in the form of a rubber plaster, forms an efficient application, and may be used when the disease is limited to well-defined patches. Occasionally, when the disease is unusually rebellious, remedies such as will excite considerable inflammation in the affected part may be employed. Such reme-

FIG. 5.



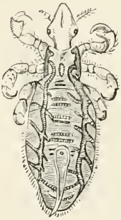
A BURROW, OR CUNICULUS.—Greatly enlarged. (After Kaposi.)

FIG. 7.



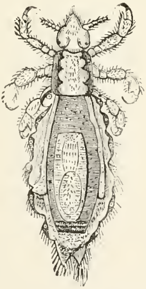
OVA, OR NITS, OF THE HEAD-LOUSE, ATTACHED TO THE HAIR.—Greatly enlarged. (After Kaposi.)

FIG. 6.



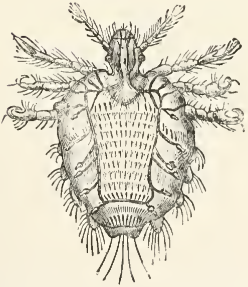
PEDICULUS CAPITIS, MALE.—× about 15. (After Küchenmeister.)

FIG. 8.



PEDICULUS CORPORIS, FEMALE.—× about 15. (After Küchenmeister.)

FIG. 9.



PEDICULUS PUBIS.—× about 35. (After Schmarda.)

dies, however, are not without danger, and should be employed only under careful supervision. Croton oil, pure or diluted with two or three parts of olive oil, may be used for this purpose, the precaution being observed never to apply it over a large surface at one time. Several such applications may be necessary to produce inflammation sufficient to destroy the fungus. Acetic acid and cantharidal collodion may be similarly employed. Permanent baldness may follow the use of such active remedies, and their employment therefore is to be recommended in rare instances only. After four to six weeks' treatment, all remedial applications should be suspended for a short time, in order that the exact conditions may again be carefully ascertained. Upon the discovery of scaliness or broken hairs or stumps or the detection of the fungus by microscopic examination, treatment should be resumed, and so on until all traces of the disease have disappeared. While local treatment is alone demanded for the cure, the influence of such applications is seemingly heightened, especially in strumous and debilitated patients, by the conjoint administration of cod-liver oil and similar nutritive tonics.

SCABIES.

Definition and Symptoms.—Scabies, or itch, is a contagious disease of the skin, due to the invasion of an animal parasite, the acarus scabiei. The presence of the parasite within the cutaneous structures excites varying degrees of irritation, and in consequence the formation of vesicles, papules, and pustules, accompanied with more or less intense itching. Secondarily crusting, and at times a mild or severe degree of dermatitis, may be brought about. The impregnated female acarus penetrates the epidermis obliquely to the rete and then horizontally, forming a minute passage or burrow, in which are deposited ten or fifteen ova. These burrows, or cuniculi, are found most abundantly in the interdigital spaces, on the flexor surfaces of the wrists, about the mammæ in the female, and on the shaft of the penis in the male, and present themselves as tortuous, straight, or zigzag, dotted, slightly-elevated, grayish or dark-gray lines, varying in length from usually two millimetres to two centimetres. The mite, or acarus (or sarcoptes hominis), is to be found at the blind end of the burrow, usually the most elevated point. Several such burrows may be found in a single case, but they are never present in great numbers, as the irritation caused by the penetration of the mite leads either to violent scratching and their destruction, or to the formation of vesicles or pustules, or to both, and their further formation is prevented. (See Figs. 4 and 5.)

The eruption due to the invasion of the itch mite is therefore, as may be inferred from the above, to be found principally in protected situations or where the skin is thin and delicate, as between the fingers, on the wrists and forearms, in the folds of the axillæ, on the abdomen, on the buttocks, about the genitalia, and in the mammary region in females. In infants and young children, especially in well-advanced cases, the scalp and face may also be involved. In children, likewise, the skin being more tender, the type of the lesions is apt to be much more inflammatory, and hence the pustular lesions are usually much more abundant, than in adults. In those predisposed, a true eczema may arise, and then, in addition to the characteristic lesions of scabies, eczematous symptoms are superadded; in chronic cases, indeed, the burrows and other consequent lesions may be more or less completely masked by the eczematous inflammation, and the true nature of the disease greatly obscured.

Scabies is a local disease, dependent solely upon the presence of the acarus. The general health is not affected, except indirectly by loss of sleep through the intense itching. It is contagious to a marked degree. It is most commonly contracted by sleeping with those affected or by occupying a bed in which an affected person has recently slept. It occurs, for obvious reasons, oftener among the poor, although it is not uncommon among the wealthier classes. The female acarus, which alone penetrates the skin, is a small, almost microscopic animal, oval in shape, the dorsal surface being convex and marked by shallow transverse furrows and provided with a number of small spikes projecting backward; on the ventral surface, which is slightly flattened, are found four pairs of legs, the two anterior pairs of which are provided with suckers, while the posterior pairs terminate in long hairs. The male acarus, which is only about one-half the size of the female, exists in comparatively small numbers, and apparently takes but little part in the production of the cutaneous symptoms.

Diagnosis.—The diagnosis in uncomplicated cases is made without difficulty. The burrows, which are pathognomonic, may usually be found upon careful examination. They should be looked for especially between the fingers and on the flexor surface of the wrists. But, apart from the presence of the cuniculi, the distribution of the eruption is, as a rule, sufficiently characteristic. An eruption of multiform lesions occurring on the hands and wrists, on the flexor surface of the forearms, in the axillary folds, about the buttocks and genitalia, and not infrequently about the feet and toes, attended with more or less intense itching, and with a progressive history, points unmistakably to scabies. It bears most resemblance to vesicular and pustular eczema, and to pediculosis. From eczema it may be differentiated by the peculiar distribution, the absence of any tendency to become confluent, and the polymorphous character of the lesions, to which is usually added a history of contagion, and progressive development. It should not be forgotten, however, that scabies may give rise to a veritable eczema in those having a predisposition to this malady. In such cases the finding of the burrows and the localization will reveal the existing cause of the eruption. Pediculosis differs from scabies in the fact that the eruption is to be found chiefly upon parts of the body with which the clothing lies closely in contact, as for instance around the neck, around the waist, etc. Moreover, as the pediculi live in the clothing, necessarily only covered portions show their irritating effects, and the hands, which are usually the first to be attacked in scabies and usually most markedly involved, are entirely free in pediculosis.

Treatment.—The disease is readily cured. As soon as the acari and their ova are destroyed, the itching and the secondary symptoms rapidly disappear. Treatment should be preceded by a soap-and-hot-water bath, immediately after which the first remedial application is to be made. There are several remedies which may be used with satisfactory results, but sulphur is perhaps the most commonly employed, and is, upon the whole, the best. It is applied in ointment form, one to three drachms to the ounce of lard, or lard and petrolatum. The peculiar sulphur odor which develops and its occasional irritating effects are its objectionable qualities. In young children, and especially in those cases in which there is much dermatitis, not more than one-half to one drachm to the ounce should be employed. Balsam of Peru is a parasiticide of some value, and in combination with sulphur may always be used with confidence. The following formula will be found exceedingly useful, especially in young children and in the highly inflammatory cases: ℞ Sulphur. præcip., ʒi; balsam. Peruv., ʒi; adipis, ℥i. In the warm season it will be necessary to add some simple cerate to this in order that the resulting ointment may be of proper consistence for comfortable use. Styrax is also a remedy of value, without the irritating effects of sulphur, and may be used either as an ointment in the strength of one part to two or three parts of lard, or pure with two drachms of alcohol and one drachm of olive oil to the ounce. Naphthol, twenty to sixty grains to the ounce, has been highly recommended by Kaposi. It is in my experience not without value, having the advantage of being colorless and odorless. In some cases, however, it is not wholly unirritating. After the preliminary soap-and-hot-water bath, the remedy selected is to be well rubbed over the whole surface. If the scalp and face are not involved, these parts may be spared in the treatment. The application is to be made morning and evening for at least two days, and better for three, and on the following day another soap-and-hot-water bath is to be taken. The underwear and bed-linen are then to be changed. In ordinary cases one such course will suffice to bring about a cure. In some instances, however, and more especially with ignorant and careless patients, and probably by reason of neglect of the details and thoroughness in making the applications, some parasites and ova escape destruction, and consequently itching will again begin to show itself at the end of a week or ten days, and a repetition of the treatment becomes necessary. After such a course, however, it is always well to wait several days at least. The secondary dermatitis which is always present in severe cases seldom requires special treatment. When it is unusually persistent or severe, soothing lotions or ointments, such as are employed in the acute and subacute forms of eczema, should be prescribed. In the case of small children and infants, as already intimated, strong applications should never be employed. A dermatitis due to too active and prolonged treatment is, indeed, often mistaken for the persistence of the scabies, and, in consequence of this error, is kept up by irritant remedies long after the destruction of the parasites.

PEDICULOSIS.

Pediculosis, phtheiriasis, or lousiness, is a contagious affection, due to the presence of an animal parasite, the pediculus, and presenting three varieties, named, according to location, pediculosis capitis, pediculosis corporis, and pediculosis pubis,—the parasite in each being a distinct species of pediculus. The first-named variety is that usually observed in children. Pediculosis corporis is, however, occasionally seen, and the crab-louse—pediculus pubis—is also in rare instances met with in the young, seated upon the edges of the eyelids and upon the eyebrows.

PEDICULOSIS CAPITIS occurs much more frequently in children than in adults. It is characterized by marked itching, and the formation of various inflammatory and secondary lesions, such as papules, pustules, and excoriations. These lesions result from the irritation produced by the parasites, and from the scratching to which the intense pruritus gives rise. The serum which exudes from the excoriated surfaces, together with the pus from the ruptured pustules, produces more or less crusting and matting of the hair. In fact, an eczematous condition of the pustular type is soon brought about. As a consequence of the cutaneous irritation the neighboring cervical lymphatic glands may become inflamed and swollen, and in rare cases may suppurate. The occipital region is the part of the scalp which is usually most profusely infested. In children with delicate skins, scattered papules, vesico-papules, and pustules and excoriations may often be seen upon the forehead and neck. In addition to the pediculi, which, as a rule, may be readily found, their ova or "nits" may always be seen upon the shaft of the hairs, quite firmly attached. These latter are dirty-white or grayish-looking, pear-shaped bodies, visible to the naked eye, and fastened upon the shaft of the hair with the smaller end towards the root. (See Figs. 6 and 7.)

The diagnosis is readily made, as the pediculi are usually to be found without difficulty, and, even when they exist in small number and are not readily discovered, the presence of the ova will indicate the nature of the affection. Pustular eruptions upon the scalp, especially posteriorly, should always arouse a suspicion of pediculosis. The possibility of the pediculosis being secondary to an eczema must not be forgotten.

Treatment consists in the application of some remedy destructive to the pediculi and their ova. Petroleum is one of the most effective remedies at command, one or two thorough applications being usually sufficient. In order to lessen its inflammability and also to mask its somewhat disagreeable odor, it may be mixed with an equal part of olive oil and a small quantity of the balsam of Peru added. The whole scalp should be thoroughly saturated one or two evenings just before retiring, and the parts enveloped with a cap or a bandage, and the remedy allowed to act overnight, to be followed the next morning with a soap-and-hot-water washing. Care should be taken not to allow the petroleum to run over the forehead or down the neck. Tincture of cocculus Indicus, pure or diluted, may also be applied, with good results. In those cases in which there is but slight inflammation of the scalp, a solution of corrosive sublimate, two or three grains to the ounce, may be employed. When the parts are decidedly eczematous, or when numerous excoriations are present, ointments are perhaps preferable. An ointment of ammoniated mercury, thirty to sixty grains to the ounce, or staphisagria, one or two drachms to the ounce, may be used. In order to remove the ova from the hairs, acid or alkaline lotions may be employed, such as dilute acetic acid or vinegar, or solutions of carbonate of sodium or borax.

PEDICULOSIS CORPORIS is dependent upon the presence of the pediculus corporis, which is larger than the variety infesting the scalp. The parasites live in the clothing, and are to be found chiefly in the folds and seams, and only exceptionally upon the skin, which they visit for the purpose of feeding. The minute hemorrhagic puncta showing the points at which the pediculi have been sucking, and the consequent papules and other inflammatory lesions, together with the excoriations, are, therefore, to be found most abundantly on those parts of the body with which the clothing comes closely in contact, as, for instance, around the neck, across the shoulders, around the waist, etc. Pediculosis corporis is, as already remarked, not at all common in children. For its treatment the clothing and bed-coverings are to be thoroughly baked or boiled, the pediculi and ova being in this manner destroyed. (See Fig. 8.)

PEDICULOSIS PUBIS is usually and typically seen about the hairy parts of the genitalia, but for evident reasons is not seen in this region in children. This parasite—the pediculus pubis, or crab-louse—is, however, occasionally met with in the young, infesting the eyelids and the eyebrows. The pediculi are rarely present in numbers, but one or several may be detected upon close examination firmly seated on the edge of the lids or on the eyebrow at the root of the hair. (See Fig. 9.) The ova may be readily discovered attached to the hairs. The excrement, appearing as minute reddish particles resembling specks of iron rust, may also be seen among the hairs and on the skin immediately below the infested part. Variable degrees of irritation result from their presence. For their destruction citrine or ammoniated mercury ointment, weakened with two to four parts of lard, may be carefully used. Frequent washing of the parts will also be of material aid.

PART II.

CONSTITUTIONAL DISEASES, AND DISEASES OF NUTRITION.

SCROFULOSIS.

By HENRY ASHBY, M.D., M.R.C.P.

For upwards of two thousand years swellings in the neck have been recognized in the human subject and known by the names of *χοιράδες*, *scrofula*, or *struma*. The origin of these terms is somewhat doubtful, and it seems to have been in dispute both by ancient and modern writers. Thus, Paulus Ægineta says, "The chœras, or scrofula, is an indurated gland, mostly forming in the neck, armpits, and groins, deriving its name either from a Greek word signifying a species of rock, or from swine, because they are fruitful animals, or because swine have swellings of the neck."[1]

It seems clear that the terms are derived from *χοῖρος* or *scrofa*, a "pig;" but what the connection was between a pig and scrofula in the eyes of the ancients it is difficult to say. Both Galen and Celsus use the word *struma* as signifying swellings or tumors in the neck, none of the ancient writers using the term in the wider signification of modern times.

The greater part of the writings of the ancients concerning scrofula are occupied in describing the treatment to be adopted for these swellings: this consisted in the application of various ointments and in giving directions for their removal by excision. Celsus describes these swellings accurately when he represents them as "indolent affections of the glands, which come slowly to maturity and prove very troublesome to the physician." This has certainly been the experience of many physicians since the time of Celsus.

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[1] Paulus Ægineta, translated by F. Adams, Sydenham Society, London, 1844.

These writings afford abundant evidence that scrofula was a common disease in ancient times, and that its principal characters were pretty much what they are at present. That scrofulous swellings were also common in mediæval times is also certain; of this there is indubitable record in the accounts preserved of the "treatment by the touch," which was practised for many centuries by the kings of England and France. Hence the name of king's evil. In England the practice prevailed as early as the times of Edward the Confessor and as recently as the reign of Queen Anne.[1] It is said that Charles II. "touched" ninety-two thousand one hundred and seven persons for the king's evil in twelve years, and, as travelling in those days was difficult and costly, applicants for this method of cure would be drawn very largely from the metropolitan districts. It seems probable from these figures that scrofulous swellings must have been exceedingly common, and Phillips is justified in his inference that scrofula was commoner in those times than in our own. What was the origin of the superstition, and how it could have been so long practised without discredit, is a marvel to us at the present time.

It is only within the last hundred years that the terms scrofula and struma came to have a wider signification. Post-mortem examinations showed that caseous glands existed in other parts of the body besides the neck, and were often associated with caseous degeneration in other organs; moreover, it was noted that those who suffered from scrofulous swellings suffered frequently also from various superficially-placed inflammations, and the opinion gained ground that the cervical swellings were only part of a more general disease; that in some individuals there existed a tendency to various catarrhs and inflammations as well as to caseous glands, and it was to this tendency in the individual that the term strumous or the scrofulous diathesis was applied. Thus there was a strumous ophthalmia, a strumous bronchitis, a strumous eczema; and, moreover, inasmuch as scrofulous glands were found caseating, the term scrofulous was applied to all caseating processes; hence there was a scrofulous hip-joint disease, scrofulous tumors of the brain, a scrofulous testicle, and scrofulous pneumonia. It was thus that the word strumous came in the last generation to have a very wide signification, and to be applied to various constitutional states which have more recently been differentiated. Thus, neither new growths, rickets, nor syphilis are looked upon at the present time as having any relation to scrofula. Indeed, the tendency at the present day among recent writers—on account of the abuse of the word strumous—is to do away with it altogether, scrofulosis being denied its position as a "self-standing" disease,—the caseating processes being looked upon as tubercular and the superficial inflammations being simply inveterate in character because they occur in children of low vitality.

What, then, do we understand when we say a child is scrofulous? or are we to avoid the use of the term because it has been used as a convenient cloak for ignorance and has been undoubtedly misused? In the first place, it is, I think, wise to use it only in a clinical sense, and not attempt, in the present state of our knowledge, to give it any definite pathological meaning, or assert that there is a definite disease to be called scrofula, as there is one called syphilis or rickets, apart from tuberculosis. It seems to me that to call certain lesions of the skin "scrofulides," as certain eruptions occurring in the course of syphilis are called "syphilides," is to stamp them as manifestations of a specific disease without just reason, and can only lead to confusion in our nomenclature.

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[1] See Phillips, Scrofula and its Treatment, p. 259.

Definition.—We may say a child is strumous who suffers from inflammations of a peculiar type, especially affecting the skin, mucous membrane, lymphatic system, bones, and joints. The distinctive characters of these inflammations are that they are induced by slight irritation or injury, are very inveterate and slow to heal, and are exceedingly apt to involve the neighboring lymphatic glands. There is a marked tendency to caseation and chronic suppuration. The tendency to these forms of inflammations has been called the scrofulous or strumous diathesis. It is wise in framing a definition of scrofulous process to avoid all reference to morbid anatomy, and to depend entirely upon clinical characters. Many of the lesions present in scrofula are unquestionably tubercular, inasmuch as it has been clearly demonstrated that the bacillus of Koch is present, and, moreover, inoculation of the guinea-pig or rabbit with material taken from such gives rise to true miliary tuberculosis. Other scrofulous lesions, as impetigo, are certainly not tubercular processes, and histologically are indistinguishable from simple eczema or similar lesions in children who are not scrofulous. The distinctive characters of scrofulous lesions are clinical,—such as their chronicity, their tendency to spread locally, and their tendency to caseate and involve lymphatic tissue.

Etiology of Scrofula.—Scrofula may be derived by inheritance or it may be acquired. In the worst forms there are both an hereditary disposition and conditions favorable for its development.

Scrofula in Parents.—Parents who have suffered from scrofula when children are exceedingly likely to have children who suffer in a similar way. It is no uncommon thing for a mother to bring her infant or child with caseous or suppurating cervical glands, having herself marks or scars in the neck of old glandular mischief; or the mother or father may have suffered from spinal caries, strumous joints, or other manifestations of scrofula.

Phthisis in Parents.—Phthisis in the parents is a common cause of scrofula in their children. It frequently happens that a man becomes phthisical, perhaps suffers from chronic phthisis for several years; the children he begets during the period of ill health show signs of scrofula, while the others are healthy.

Syphilis.—Syphilitic parents not unfrequently beget children who become scrofulous. Children the victims of hereditary syphilis, and who suffer or who have suffered from interstitial keratitis or who bear the marks of past syphilitic lesions, not infrequently have also caseous cervical glands. It may happen in a family that the older children suffer from hereditary syphilis, whereas the younger are simply scrofulous, suffering from eczema, phlyctenular ophthalmia, caseous glands, etc. It would seem as if the virus of syphilis may become attenuated and simply produce an enfeebled constitution which shows itself in scrofulous lesions.

Consanguinity, etc.—Some writers, such as Rilliet, Lugol, Comby, have attributed scrofula in children in some instances to consanguinity on the part of the parents; in other cases to the youth of the parents. Comby lays especial stress on the latter, and quotes instances where the parents were nineteen and seventeen years of age or thereabouts, their first-born being scrofulous and their later children healthy.

Advanced age of the parents, as well as extreme youth, has been said to predispose to scrofula. It is probable that in both cases the infants may be weakly, and, if the conditions of life are favorable thereto, they readily become scrofulous.

Rapid childbearing also apparently predisposes to scrofula.

Effects of Food.—That insufficient or improper food tends to produce scrofula, where an hereditary tendency exists, cannot be doubted, but whether it will produce it in the absence of any hereditary taint cannot be stated with certainty.[1] In most cases where children have been insufficiently fed, as, for instance, in the orphan asylums and workhouse schools of a generation or two back, there was generally overcrowding and bad air, and the inmates of such asylums were, many of them at least, the subjects of an hereditary disposition to scrofula. There cannot be a doubt, however, that with the more liberal diet that has been in vogue during the last few years in workhouse schools, as well as in the homes of the poorer classes, there has been a marked diminution in the amount of scrofula.

A realistic picture of the condition of things which existed in the House of Industry in Dublin has been given by Carmichael in his lectures. He says, "Some years ago I had a very melancholy but convincing proof of the effects of improper food in producing scrofula, upon five or six hundred children in the House of Industry, of all ages from a year to puberty. The diet of the children consisted of coarse brown bread, 'stirabout,' and buttermilk, generally sour, for breakfast and supper; of a mixture of potatoes and esculent vegetables, either cabbage or greens, for dinner; and sour buttermilk again for their drink. They were confined to their dormitories and school-rooms, of insufficient extent for their number, there being no play-ground for the children, consequently they were deprived of that exercise so natural and necessary for the development of the frames of young animals, and which might have enabled them to digest in some degree their wretched and unwholesome diet. Under this cruel mismanagement, they lost all spirit for exercise or play; and on visiting the rooms in which they were incarcerated, the air of which was impure to a degree only to be compared to jails of former times, these wretched little beings were seen squatted along the walls of their foul and noisome prison, resembling in their listless inactivity an account I have somewhere read of savages met with in Australia, their faces bloated and pale, and their stomachs, as they sat, nearly touching their chins. On a closer examination of these children, it was found that in general the upper lip was swelled, the tongue foul, or sometimes of a bright red (indicative of acidity of stomach), the breath offensive, the nostrils nearly closed by the swelling of the mucous membrane, the abdomen tumid and tense, and the skin dry and harsh; but, that which most appertains to my present subject, the cervical glands were more or less swelled and tender; and I am within bounds when I assert that nearly one-half of these unhappy children had the characteristic signs of scrofula in their necks."

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[1] That scrofula can be acquired in the absence of hereditary taint is rendered probable by the fact that wild animals, such as lions and monkeys, become scrofulous in captivity.

The effects of insufficient food, more especially in those cases where there had been a deficiency in meat and fat and an excess of vegetable and farinaceous food, were apparent in the amount of scrofula which prevailed among the English and Irish rural population a generation ago, and which prevails to a certain extent at the present time. The average weekly wage of an English agricultural laborer some years ago was ten or eleven shillings (two and a half dollars) per week. Out of this, rent of cottage had to be paid and a family supported. The dietary largely consisted of bread, potatoes, cheese, tea, and small beer; butcher's meat was unknown, and bacon, butter, and milk were rare luxuries. With this dietary scrofula was exceedingly common,—more common than in the large manufacturing towns, where, with all the unhealthy surroundings and vitiated atmosphere, wages were better than in the country, and consequently a better dietary prevailed.

The same kind of evidence in support of insufficient food producing scrofula is afforded by the condition of English prisons some few years back. It was shown that persons entered these establishments in perfect health and free from hereditary taint of scrofula or tuberculosis; but they developed enlarged glands or other manifestations of scrofula during their stay. At the present time, with an improved dietary in English prisons and better ventilation, they are stated to be the healthiest places in the country, and prisoners of the lower classes are mostly discharged in a better state of health than when they were committed.

Bad Hygiene and Unhealthy Surroundings.—Vitiated air from overcrowding, absence of sunlight, exposure to cold and damp, are important factors in producing acquired scrofula. It is difficult or impossible to estimate the effects of one or the other by itself, as they so constantly are associated together, being the conditions usually accompanying squalor and poverty. The importance of overcrowding as a means of giving rise to the manifestations of scrofula cannot be overestimated, for not only does breathing air vitiated with human breath lower the general health of the body, but, if the bacillus of tubercle is the active agent in producing scrofula and tuberculosis, the crowding together of infected individuals must favor the infection of those who are free.

Age.—The manifestations of scrofula are in a large measure confined to the early or later years of life. The commonest period is perhaps between three years of age and the commencement of puberty, though infants a few months old may suffer from glandular enlargements and abscesses. During childhood the lymphatic system is functionally active, and we find it in consequence more liable to take on inflammatory action than in later life. Thus we find caseation of the lymph-glands, lymphadenoma, Hodgkin's disease, commoner in childhood than in adult life.

Infection.—Is scrofula infectious? Does it spread from one child to another, or from some of the lower animals, more especially the cow, to human beings? This question resolves itself into the larger one, Is tuberculosis infectious? The older observers, who saw the fearful prevalence of scrofula in workhouse schools and similar institutions where many children were crowded together, believed that scrofula spread by infection. In a certain sense this was no doubt true. Purulent ophthalmia, impetigo, as well as the eruptions caused by pediculi, would in an overcrowded and badly-managed institution, where the health of the inmates was below par, readily pass from one child to another. Glandular enlargements would no doubt follow in those who had an inherited tendency to scrofula, or in those whose health was lowered by the effects of an improper diet and unhealthy surroundings.

Since the discovery of the possibility of the inoculation of tubercle by means of caseous material, and more especially since the discovery of the tubercular bacillus, the question of infection has assumed a new light, especially as to the possibility of infection by breathing air containing the bacilli, by means of vaccination, or by consuming milk from a tuberculous cow. Only the last two will be considered here. With regard to vaccination, it must be admitted that after vaccination with both calf-lymph and humanized lymph various glandular enlargements and chronic tubercular abscesses have made their appearance. I have seen on several occasions a few weeks after vaccination the cervical glands enlarged on the same side of the neck, a rapid spread to other, neighboring glands, followed by numerous "cold abscesses" in various parts of the body, caries of the nasal bones, and dactylitis. In these cases the vaccination was with calf-lymph, and the local irritation was more considerable than usual. The question arises, did the lymph contain tubercular bacilli? was there a local tubercular process at the seat of vaccination and then an infection of the neighboring lymphatics? It is by no means necessary to assume that this was so. We know that lymphatic glands enlarge and become caseous secondarily to all forms of skin irritation on the face or other parts which are in no sense tubercular, and it is far more probable that the process of vaccination led to an irritation of the glands, which in a scrofulous child became caseous. It is highly improbable that lymph from either a healthy calf or a healthy child should contain tubercular bacilli. With regard to the second question, —may a healthy child become scrofulous by drinking milk from a cow suffering from lung-tuberculosis or tuberculosis of the udder? It is well known that cows frequently suffer from tuberculosis; it has been stated, on the authority of veterinary surgeons, that twenty-five to fifty per cent. of beeves slaughtered in England were tubercular. Indeed, Dr. Carpenter, of Croydon, stated, at a recent meeting of the British Medical Association at Glasgow (August, 1888), that a London inspector informed him that eighty per cent. of all the meat sold in the London market bore evidence of tubercular mischief, and that if the whole of this were condemned the inhabitants of London could not be fed. It is certain that the milk of cows suffering from tuberculosis of the udder contained tubercular bacilli; it is less certain that cows suffering from general tuberculosis give tubercular milk.

Pathology.—It must early have struck pathologists what a similarity there was between the morbid anatomy of scrofula and tubercle. Whatever differences there might be clinically, the difference was ill defined in the dead-house, for caseous masses in the brain, testis, and glands were found associated with gray granulations in the lungs. It must have been noticed from the first that children who died with tabes mesenterica usually had tubercles in their lungs. The identity of scrofula and tubercular phthisis was denied by many of the older writers, such as Carmichael, who, however, admitted that the children of phthisical persons were generally scrofulous. Others, as Roche, maintained their identity, the only difference being in the seat of the deposit. This view was strongly upheld by Graves, who in his Clinical Lectures says, "The most important thing for a student to impress on his mind with regard to all cases of phthisis is, that the pectoral symptoms, of whatsoever nature they may be, are caused by scrofulous inflammation. If you trace the phenomena of external scrofulous abscesses, you will be struck with the close analogy they bear, in their manner of appearance, their progress, and termination. to the ulcerations of the lungs in phthisis. The same slowness, the same insidious latency, the same gradual solidification and gradual softening, the similarity of the puriform fluid secreted in each, the analogous occurrence of burrowing ulcers and fistulous openings, the close approximation in the form of their parietes, and the difficulty in healing remarked in both, make the resemblance between them extremely striking. Compare scrofulous inflammation of the hip and knee-joint with phthisical suppuration of the lungs: have we not the same kind of hectic fever, the same flushings and sweats, the same state of urine, the same diarrhœa, the same state of appetite, and the same emaciation?"[1]

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[1] Graves's Clinical Medicine, p. 510.

This strong resemblance between scrofulosis and tuberculosis has been well put by one of the most recent writers on scrofula, who says, "Since tubercle was first described, its fortunes and those of scrofula have been linked together. In all its changes, in all its losses, into all the false positions into which it has been thrust first by one pathologist and then by another, scrofula has had its share. Scrofula at one time posed as a tubercular process; tubercle at another time has been described as a scrofulous process. Once more the two conditions have been quite distinct, and have been even antagonistic; and lastly they have been identical, with no line of separation between them."[1]

The available evidence of the identity or difference of the two processes, the scrofulous and the tubercular, may be considered under three heads:

1. Evidence afforded by the naked-eye appearance of the organs removed post-mortem or by operations.

2. Evidence afforded by microscopical examination and cultivation of the specific bacillus.

3. Evidence afforded by inoculation-experiments on rabbits and guinea-pigs.

1. *Naked-eye Appearances.*—Examination of enlarged lymphatic glands which have been excised from the neck often shows not only caseous patches and points of softening, but also gray granulations singly or in clusters. The enlarged bronchial and mesenteric glands similarly show caseation and also gray granulations. It can hardly be held that there is any difference between the processes in progress in the cervical and bronchial glands: if one set is tubercular, the other set surely is also. That caseous bronchial glands are tubercular is shown by the way in which they inoculate the neighboring lung; caries of the bodies of the vertebræ has been known to act in a similar way, the tubercular process spreading by contact from the carious bone into an adherent lung. The former may be seen in the bodies of children who were tubercular but who have died of some intercurrent disease, or of some acute tuberculosis, as meningitis. A lung may be free from tubercle except at its root, where a caseous gland is embedded in lung-substance, the lung immediately around the caseous gland containing gray tubercle. In such cases the sequence of events has been tolerably clear: there has been some simple lung-trouble, the mediastinal and bronchial glands have become enlarged and caseous, and then there has been an infiltration or an infection of the lung surrounding the caseous glands, which spreads into the lung from the roots, and a tuberculosis of the lungs is the result. It appears to me that no stronger evidence could be produced of the identity of the scrofulous and tubercular processes. Similar evidence is obtainable from caseous mesenteric glands: a tubercular peritonitis is constantly associated with mesenteric disease, and appears to arise, in many instances at least, by direct contact.

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[1] Treves on Scrofula, p. 4.

That tuberculosis of the mesenteric or bronchial glands forms a greater standing danger to life than tuberculosis of the cervical glands is certain, but this is accounted for by their position, without the necessity of assuming that the tuberculosis is of a less intense nature in the one case than in the other. The cervical glands are surrounded by structures which do not readily become tubercular, and the glands themselves may caseate and discharge without any local spread. The risk of a local extension is very much greater when the glands are deeply seated and surrounded by important structures such as the bronchial or mesenteric glands. At the same time it is probable that caseation and suppuration of these glands take place oftener than is sometimes thought, and recovery results, as it is not uncommon to find them cretaceous at post-mortem examination or actually suppurating in cases which have died of some intercurrent disease. It is difficult to say if there is a greater tendency to suppurate in the external lymphatics when caseous than in the more deeply seated, as some have maintained; as the symptoms given by a suppurating mediastinal or mesenteric gland are necessarily more or less indefinite, and, moreover, a general spread of tubercle is likely to take place before suppuration commences.

In scrofulous or tubercular disease of the joints the tendency to a general tuberculosis is not perhaps great, and children linger long with scrofulous hip- or spine-disease without there being a general tuberculosis; but certainly tubercular meningitis is not uncommon in such cases or an acute tuberculosis, and this acute general infection is apt to follow an operation or a forcible flexion of a scrofulous joint, as my colleague Mr. G. A. Wright has had occasion to notice. It would seem as if at times a stirring up or irritation of the bone-disease led to a general infection.

2. *Microscopic Examination.*—The evidence afforded by microscopical examination of the tubercular nature of scrofulous glands and other scrofulous lesions turns principally on the presence or absence of the bacillus tuberculosis. Numerous "giant cells" are frequently present in lymphatic glands, caseous bone, and lupus, but the value of these as evidence of tubercle has been diminished of recent years by their presence having been discovered in some sarcomas and other growths. It is almost universally admitted at the present time that the presence of Koch's bacillus stamps the process as tubercular, even though gray tubercles or the histological characters of what has hitherto been regarded as tubercle are absent. The absence of the bacillus cannot, of course, be taken as evidence of the non-tubercular nature of any lesion. It is present mostly during the active growth of tubercle, and may disappear when degeneration and suppuration are present. Very many sections would have to be examined before it could be definitely stated that it was certainly absent from any structure.

Tubercular bacilli have been demonstrated in scrofulous bone, joints, synovial membrane, lupus, cold abscesses, glands, tongue, testes, uterus, and appendages. Among the observers of these are Koch, Cornil and Babes, Demme, Albrecht, and Hauck.

3. *Inoculation Experiments.*—Many experiments have been made by inoculating rabbits and guinea-pigs with caseous materials taken from various scrofulous lesions, to ascertain if a more or less general tuberculosis was set up. Rabbits and guinea-pigs have been usually selected for these experiments, on account of their known predisposition to tuberculosis. Many experimenters have repeated these experiments, with more or less success. Some of these recorded by H. Martin[1] are especially complete and important. A short summary will be given of the most important.

(1) A fragment of caseous bone, taken from a necrosed phalanx of a child of three years, was inserted into the peritoneal cavity of a guinea-pig on April 14, 1881. At the commencement of June an ulcer formed at the seat of inoculation, and on June 21 the animal died. At the autopsy the mesenteric glands were found enormously enlarged and caseous; the mesentery contained yellow tubercles, and tubercles were also present in the kidneys and spleen; the lungs and bronchial glands were also affected. A small piece of caseous mesenteric gland was introduced into the peritoneum of a second animal, which died of general tuberculosis on September 16. A third animal was inoculated from the second; this also died of general tuberculosis on October 26. In all three animals the mesenteric and bronchial glands were enormously enlarged and caseous.

(2) Some yellow crusts were taken from the scalp of a child of two and a half years suffering from eczema of the head and face, and introduced into the peritoneal cavity of a guinea-pig on May 15, 1882. The animal died in January, 1883. The result was negative, no tubercle being found.

(3) An infant of eight months, suffering from impetigo of the face and scalp, with enlarged submaxillary glands, died suddenly in convulsions. No lesion was found post mortem to explain why death had taken place; the lymphatics in the neck were much enlarged, but no caseous foci were detected. Pieces of the enlarged submaxillary glands were inserted into the peritoneal cavities of two guinea-pigs on March 5, 1883. One animal died on the following April 2; the autopsy was negative as far as tubercle was concerned, but a small abscess was present, and a slight enlargement of the mesenteric glands. A second animal was inoculated from the abscess and a mesenteric gland: this animal died eight months after; the mesenteric glands were enlarged and suppurating. A third animal was inoculated from the second, with negative results. The second animal inoculated on March 5 died in April, 1884; there was no tuberculosis.

(4) An infant of seventeen months suffering apparently from tuberculosis had impetigo of and an abscess in the scalp; the abscess was opened and some of the pus injected into the peritoneal cavity of two guinea-pigs. Tuberculosis was produced in both animals.

(5) At an autopsy made on an infant of four years, the submaxillary glands were found enlarged and congested, and in one of them a caseous

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[1] Revue de Médecine, tome iv., October, 1884, p. 786.

nodule was found. There was a caseous nodule in the right lung and also in a lymphatic gland at the hilus. During life the child had suffered from chronic ophthalmia, and there was a cicatrix the result of an old glandular abscess. Caseous matter taken from the submaxillary glands caused tuberculosis when introduced into the peritoneum of a guinea-pig.

(6) At the autopsy of an infant of four months, a nodule of caseation was found beneath the pleura, surrounded by gray granulations; there were also caseous bronchial glands. A fragment taken from the caseous nodule in the lung was introduced into the peritoneum of a guinea-pig; the animal died of a general tuberculosis. A fragment of a lymphatic gland which was simply enlarged and congested was taken from this guinea-pig and introduced into the peritoneum of a second animal; this also produced a general tuberculosis.

A series of highly-interesting inoculation-experiments were made by Eve[1] upon rabbits with material from caseous glands. These experiments were undertaken to confirm or disprove those of Arloing, who had come to the conclusion that there was a marked difference in the results in inoculating material from scrofulous glands and gray tubercle. Eve found that inoculation with the material from caseous glands produced visceral tuberculosis in rabbits and guinea-pigs, though the tuberculosis thus set up was not so rapidly fatal, as a rule, as inoculation with miliary tubercle. He found but few bacilli in strumous glands, but the bacilli were very numerous in the visceral tuberculosis in the rabbits inoculated from strumous glands. Another interesting point noted by Eve was that the bacilli in the visceral tuberculosis, when stained, showed even outlines; in the strumous glands they had beaded outlines: these observations are important when taken with those of Malassez and Vignal, who have described fine granular masses, apparently collections of spores, possibly of Koch's bacilli, in caseous glands, when the bacilli themselves were few in number or absent.

What is to be regarded as the outcome of these experiments and observations? In the first place, they point clearly to the conclusion that eczema, impetigo, chronic ophthalmia, and ozæna are not tubercular processes though they occur in a strumous child and are chronic and inveterate in character. Inoculation with the crusts of eczema or with the discharges from the nose or eyes may set up a form of septicæmia, but not a tuberculosis; such discharges do not contain Koch's bacillus. In opposition to this, however, it may be stated that Volkmann, at least on one occasion, reports the presence of tubercular bacilli in some crusts of impetigo; and Unna regards some forms of impetigo as true tuberculosis of the skin. In the next place, it is also clear that the first stage of glandular enlargement prior to caseation is not tubercular, as it is only when caseation begins that tubercular bacilli are present and inoculation-experiments succeed. It must, however, be remembered that caseation is a gradual process, and it may be impossible to determine by appearance alone when a tubercular process commences. The smallest caseous foci appear to be sufficient to indicate the presence of tuberculosis.

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[1] See British Medical Journal, April 14, 1888.

We may have, then, as the order of events:

1. An impetigo or ozæna or irritation caused by dentition,—non-tubercular.
2. Secondary enlargement of lymph-glands,—non-tubercular.
3. Caseation of lymph-glands,—tubercular.

From this it would appear that the bacillus of tubercle in some way or other finds an entrance to the inflamed gland and starts a tubercular process. How the tubercular bacilli find an entrance into the system is uncertain. Some have supposed that in some cases the bacilli have been derived from the child's parents, and have lain dormant in the system till favorable conditions for their cultivation occur. Others, like Unna, believe that an impetigo is really a skin-tuberculosis; the skin having become denuded, the bacilli have found entrance into the system from without, set up a local process, and passed on into the lymph-glands. That this is not impossible must be admitted, but it will not explain how enlarged glands following cutting teeth or due to the irritation of a carious tooth become tubercular. Or a child has a fall on its hip or spine; there is no external wound; a slight inflammation is set up in the joint or the epiphyses, and a caseation follows: in this case there can be no question of the entrance of the bacilli immediately from without. Or, to take another instance, a child has a fall on the back of his head, begins to have cerebral symptoms and optic neuritis some months after, and dies eventually with a caseous mass in his cerebellum. We can only suppose that the fall gave rise to some local hemorrhage or was followed by some inflammation of the cerebellar substance, which, as in the injured hip, became tubercular. How the bacilli find entrance into the system can only be conjectured: presumably they enter with the breath or in the food, and find their way in the blood-current to the glands or other part. They seem to be able to start a tubercular process in those parts only which are in an inflammatory or unhealthy condition. Thus, congested or chronically-inflamed glands, whether externally placed or situated in the mediastinum or mesentery, are apt to become tubercular. A chronic broncho-pneumonia or a joint which is chronically inflamed seems to form a suitable nidus for the bacilli, and a tuberculosis succeeds a simple chronic inflammation. It is this tendency for a simple inflammation to become tubercular which distinguishes the scrofulous diathesis.

Morbid Anatomy.—The naked-eye appearances presented by scrofulous glands vary. In an early stage, before any tubercles or caseation have made their appearance, the enlarged gland appears paler than normal and is usually of a somewhat softer consistency. This paler appearance is due to an increase in the number of lymph-corpuscles, both in the lymph-paths and in the gland-substance. The lymph-paths appear to be crowded with leucocytes; the gland-substance and the fibrous capsule and trabeculæ are infiltrated with them. Later patches near the central portion of the gland make their appearance, which are paler than the rest of the gland; these afterwards become distinctly yellow in color and in point of fact are the spots where the caseation commences. In some cases the first change noted consists in the appearance of gray granulations. The patches of caseation join together, so that in time the whole gland may caseate. In a more advanced state the caseous material may soften into pus, so that the central portion of the gland becomes converted into an abscess. While these inflammatory changes are proceeding within the gland, more or less periglandular inflammation takes place, so that the gland mostly becomes fixed to the surrounding structures: thus a bronchial gland may become adherent to a bronchus, or the cervical glands to the muscles or fascia. The caseous glands do not necessarily suppurate, but undergo various retrograde changes: fibroid tissue forms, so that the capsule becomes thickened, and the gland itself shrinks and is more or less fibrous on section. The caseous material may dry up and the gland become cretaceous. The rapidity with which caseation and suppuration take place varies very considerably: in some cases the enlargement takes place quickly, followed by an abscess; in other cases there may be a quiescent stage which may last for many months or years. The gland may enlarge and remain so for a long time, then, perhaps as the result of an injury, or without known cause, the gland quickly softens as pus forms. The histology of scrofulous glands has been studied with much care by Treves. It has already been stated that the first change consists in an infiltration of the gland with leucocytes; at the light-colored spots, which are evidently inflammatory foci and which afterwards become caseous, an active cell-division is going on, and, moreover, there are numerous larger cells with glistening protoplasm; as caseation advances, these cells gradually disappear, leaving only a fatty detritus. In other and more chronic cases there are more typical tubercles to be seen with giant cells and numerous tubercular bacilli.

Symptoms of Scrofula. PHYSIOGNOMY.—Much was made by the older writers of the physical peculiarities of those who were scrofulous, and many fanciful descriptions were given of the different types to which scrofulous individuals could be referred. There were the *sanguine* type, the *phlegmatic* type, and the *pretty* type. It cannot be said that these divisions are of any great importance in practice, for the simple reason that a diagnosis is made, in the vast majority of cases, not from any peculiar cast of face or general physiognomy, but from the symptoms which are present. It must constantly be the experience of the physician to see children suffering from enlarged glands or hip-disease, who either are the pictures of robust health, or else have the appearance of being delicate children without its being possible to refer them to any definite type. A child is "strumous"-looking, or gives one the idea that it is scrofulous, because we note at a glance a chronic ophthalmia, or an impetigo on the face, or enlarged glands in the neck; just in the same way as a child has a syphilitic look because we see it has a flattened bridge to its nose or has scarring about the mouth and pegged teeth.

The types of the older writers may be described as follows:

1. *The Sanguine Type.*—Children belonging to this class have light hair, fine delicate skins, and oval faces, are slight in build, tall, with well-formed hands and feet. The eyelashes are long and the hair fine. They are of bright and excitable dispositions.

2. *The Phlegmatic or Lymphatic Type.*—Children belonging to this class have coarse and irregular features, large jaws, prominent malar bones; the nose is thick, the upper lip swollen, and the ears large. The skin is coarse, with thick subcutaneous tissues. Such children are apathetic and slow in their movements; they suffer from cold hands and feet.

3. *The Pretty Type.*—This type is represented by individuals who have some of the characteristics of both the other two. The general features may be those of the phlegmatic type, with many of its features absent or conforming to those of the sanguine type.

The practical point in connection with these types is, that the sanguine type is the most common form when the disease is due to heredity, while the phlegmatic type is usually associated with the acquired form of disease or where the child has been under influences which have tended to develop the disease in those cases where there has been an hereditary taint. "Parochial scrofula," the form most commonly seen in workhouse schools and penitentiaries, is of this type.

General Manifestations of Scrofula.—It is impossible to make any definite assertion which shall be generally true as to what is the first manifestation of scrofula. Bazin has attempted to do this by dividing scrofula into three or four periods, after the manner of syphilis: thus, he would include the superficial lesions, such as eczema or lupus, in his primary period, glandular enlargements in his second, affections of bones and joints in his third, and tubercular disease of the lungs in his fourth period. It is more than doubtful if these divisions aid the attempt to simplify the description of scrofulous manifestations, inasmuch as so many exceptions must be mentioned and so many irregularities noted as to deprive the classification of its value.

The early manifestations may include eczema, glandular enlargement, hip-disease, cheesy tumor of the cerebellum, and are by no means confined to lesions on the surface. Sometimes we have to deal with a conjunctivitis or ophthalmia, or the first symptoms may be those of a chronic intestinal catarrh followed by disease of the mesenteric glands.

It will be most convenient, instead of speaking of stages or periods, to give a detailed description of the various scrofulous affections according to the tissues or structures involved.

SKIN-AFFECTIONS.—The principal skin-affections which are characteristic of scrofulosis are eczema, lichen, lupus, and cold abscesses.

Eczema is exceedingly common in scrofulous children: the skins of such seem very readily to take on inflammation. It is especially apt to occur about the nose and lips, ears and scalp; in the former situations the secretions from the nasal mucous membrane and mouth are often the exciting cause. In the same way a chronic discharge from the ear may give rise to an eczema of the meatus and surrounding parts in consequence of the irritating nature of the discharge. Very often a scrofulous eczema presents no peculiarity which will distinguish it from an eczema in an otherwise healthy child; usually, however, there is a tendency to pus-formation; instead of serum exuding from the vesicles, cloudy semi-purulent fluid oozes out, which as it dries forms yellow crusts; this is especially so in anæmic unhealthy children. A similar form of eczema may in scrofulous children be present at the seat of vaccination, after the vesicles have dried up. An eczema is often the starting-point, or rather perhaps marks the commencement, of scrofulous manifestations. A few vesicles may appear on the scalp of an infant a few months old, these spread and crusts form, and the skin becomes infiltrated, the lymphatics enlarge, or perhaps small cold abscesses make their appearance in different parts of the body. In some cases superficial abscesses form in the scalp after an eczema; they are mostly painless and not accompanied by much inflammation.

The eczemas or impetiginous eruptions so common about the mouth and nose of weakly children are fertile sources of glandular enlargement. They are very chronic and difficult to cure so long as there is any discharge from the nose.

Lichen Scrofulosus.—This is a form of lichen which is so named because it occurs most commonly in children who exhibit evidence of scrofula elsewhere: thus, it is perhaps associated with enlarged glands or chronic joint-disease. The papules are usually small,—not larger than a pin's head; at first they are bright red, but gradually fade, becoming pigmented, so that they assume a pale-brownish color. The papules tend to arrange themselves in circles or segments of circles. In rare cases the whole body is affected, but usually the favorite seats are on the sides of the chest and flanks, the neck and limbs being less often affected (Crocker). It is remarkable that, unlike most forms of lichen, there is little or no itching. They are exceedingly chronic in their course; they undergo desquamation and gradually disappear without discharging their contents, simply leaving a stain. The eruption may last for years. The diagnosis is not usually difficult, as some other evidence of scrofula is usually present; a difficulty may occur in confusing syphilitic lichenous rashes with this form, as the characters of the rash are very similar (Crocker). One must depend for a diagnosis on the history or other evidences of syphilis.

Lupus; Scrofulide Tuberculeuse.—Lupus may be, and as a matter of fact often is, the sole evidence of scrofula present in an individual. A history, however, of phthisis or of caseous glands may usually be obtained in the family, perhaps in one or other of the parents of the patient. It mostly begins in early life: the commonest time perhaps is between the ages of three years and twelve years. The commonest seat is the face, especially on the cheek or nose, but it may occur on the backs of the hands, elbows, knees, or indeed in any situation in the body. It first appears as a collection of small spots or papules of a dullish-red color, gradually developing into tubercles of a brownish or fawn color. After a time these groups of papules coalesce to form a dull-red patch of indurated tissue with a desquamating surface. This patch extends by the formation and coalescence of tubercles along its edges, while a cicatrizing process is going on nearer the centre. The course is exceedingly chronic, going on for years, advancing irregularly in one direction while cicatrization is going on in another part. It is exceedingly curious that the lymph-glands rarely become implicated. Both histologically and pathologically lupus closely resembles the undoubtedly tubercular or scrofulous lesions. Bacilli undistinguishable from tubercular bacilli, and giant cells, are present.

Scrofulous Gumma, Cold Abscess.—Small subcutaneous nodules, which soften and form abscesses, may make their appearance in infants a few months old; usually they first appear during the first four or five years of life. They are frequently associated with eczema, caseous glands, or dactylitis. When first observed they consist of small subcutaneous nodules the size of a small pea or less, mostly situated on the limbs or trunk, and readily movable beneath the surface of the skin. In some cases they are very numerous, almost giving the idea of a multiple infection having taken place, the nodules being scattered over the body and limbs. The size they attain before softening varies considerably: they may never become larger than peas, thin pus eventually discharging through a minute hole in the skin; or they may be much larger, perhaps, their contents amounting to a drachm or more. They are mostly very chronic in their course, very slowly suppurating, the skin gradually assuming a reddish-purple color and becoming thinned till at last the pus, often mixed with blood, escapes. Then not infrequently a so-called scrofulous ulcer forms.

That these caseous nodules are tubercular in nature has been demonstrated over and over again, as the abscess-wall displays a tubercular structure and bacilli are often present, though not always in large numbers, in the discharge.

Scrofuloderma.—This term is applied to a form of dermatitis which is common in strumous children in connection with caseous glands or cold abscesses. In the commonest form the skin over caseating glands becomes reddened and flabby, and the dermatitis may spread beyond the limits of the glands over the face and neck. The dermatitis may be independent of glands, beginning as tubercles in the subcutaneous tissues, which gradually break down, the skin becoming red, indurated, and riddled with sinuses.

Ophthalmic Affections.—Affections of the eye are not infrequent in the scrofulous. They are mostly superficially seated; they give rise to much irritation and photophobia; like most scrofulous affections, they are very intractable and apt to relapse. They do not, as a rule, lead to serious damage. The principal affections are as follows:

1. *Phlyctenular Ophthalmia.*—Small papules termed phlyctenulæ make their appearance on the "white of the eye" near the edge of the cornea, or they may be seated on the cornea near its margin. There is usually more or less conjunctivitis and intolerance of light.

2. *Corneal Ulcers.*—Minute ulcers or abrasions of the corneal substance, situated at or near the centre of the cornea, are apt to occur; there is more or less attendant congestion of the conjunctiva, with pain and intolerance of light. Corneal ulcers are often very chronic, and leave behind a small opacity or milk spot which remains for months or even years.

3. *Tinea Tarsi.*—An acute ophthalmia is apt to leave behind a chronic inflammation of the edges of the eyelids, the eyelash follicles and glands being the actual seat of the disease. There is an excessive secretion which glues the eyelashes together during sleep, while excoriations are apt to appear and scabbing take place. The inflammation, if it continues for a long time, may produce permanent loss or stunting of the eyelashes or a turning in or eversion of the stunted eyelashes.

"*Styes,*" or suppurative inflammation of the eyelids, are common among the scrofulous.

Acute inflammatory affections of the conjunctiva are also common, especially where a number of children are congregated together.

OTORRHŒA.—Catarrhal inflammation of the middle ear is very frequent in strumous children, and is often associated with catarrh of the Eustachian tube and fauces. Deafness more or less temporary may be produced. In a later stage the petrous portion of the temporal bone may become affected and the discharge purulent; the membrana tympani is perforated. There is a chronic discharge of pus from the ear, with perhaps eczema of the auricle. Otorrhœa may be secondary to some acute disease.

MUCOUS MEMBRANES.—Chronic catarrh of various mucous membranes of the body takes place with great frequency in scrofulous children; the most characteristic of these are chronic tonsillar enlargement, post-nasal adenoids, chronic catarrh of the nasal mucous membrane, and also of the membrane lining the vagina.

Chronic tonsillar enlargement may occur in infants under a year, and may be the first symptom of a tendency in the direction of scrofula; in perhaps the greater number of cases it is only when the child is two or three years old that any decided hypertrophy is noticed. It is important to bear in mind that enlargement of the tonsils may be present and give rise to no very definite symptoms in young children. The hypertrophy of the lymphatic tissue is not necessarily confined to the tonsils; indeed, the tonsils may be of normal size and yet the mucous membrane of the pharynx and nasal tract with the adenoid tissue present may be thickened and congested. There may be the "post-nasal adenoids" or a "diffuse hypertrophy of the tonsils;" the nasal mucous membrane is frequently affected; there is thickening, perhaps ulceration, and constant stuffiness about the nose, with an excessive discharge of mucus. In the worst cases the nasal discharge "frets" the skin of the upper lip and an irritation eczema results. This chronic catarrh of the nasal mucous membrane with soreness at the anterior nares is one of the most frequent sources of enlargement of the cervical glands.

Vaginitis, Catarrhal Vulvitis.—A catarrhal state of the mucous membrane lining the vulva, vagina, and more or less the urethra is common in strumous girls of two to seven years of age. The first thing to call attention is the presence of pus or blood on the child's linen, or perhaps she is noticed to scratch, especially if, as is often the case, thread-worms are present either in the vagina or in the rectum. An examination shows that an excessive quantity of mucus or muco-pus is discharged from the mucous membrane of the vagina external to the hymen, and the urethral mucous membrane may also be affected, though perhaps in lesser degree than is usual in gonorrhœal infection. The chronic condition may follow an acute attack, though, in scrofulous children especially, the catarrh may be chronic from the first. It may arise from infection, or some irritation, such as the presence of thread-worms, may be the means of setting it up.

DISEASES OF BONE.—Diseases of bones and joints are very common among the scrofulous, and are among the most formidable affections to which they are liable. Among these are spinal caries, diseases of various joints,—hip, knee, elbow, wrist,—and caries of various of the long bones, as the phalanges of the fingers, the ribs, and the sternum. Caries of the nasal bones and the petrous portion of the temporal bone is by no means uncommon. The bone-affection which is perhaps the most common, and is certainly almost exclusively found in the strumous, is dactylitis. The phalanges of the hand or metacarpal bones are the most commonly affected. It is more especially common in young children. A phalanx or metatarsal bone is noted to be enlarged, the swelling becoming more or less of a flask-shaped appearance; after a while the swelling softens, the skin reddens and gives way, and a thin unhealthy pus escapes. In some cases the swelling gradually subsides and disappears without discharging. According to Treves, the disease commences in the centre of the bone and gradually expands it. The course of strumous dactylitis is chronic; sinuses are apt to form, and pieces of caseous material and necrotic bone, for many months.

It is unnecessary here to give any special description of spinal caries or disease of any of the joints.

LYMPHATIC GLANDS.—As already pointed out, the most characteristic lesions in scrofula are found in connection with the lymphatic glands. Some gland, more often several glands, become enlarged, and, after remaining in this condition for a more or less lengthy period, suppurate, the skin gradually becomes undermined and breaks, the broken-down glands discharge, and a sinus is formed, which eventually cicatrizes after many months, perhaps years, of chronic suppuration. In perhaps the majority of cases, a chain or cluster of glands become caseous; there is a marked tendency for one gland after another to become affected. The cervical glands are far more frequently affected than the glands in other regions.

The *exciting causes* of the glandular enlargement are very diverse: in the large majority of cases it is the result of some form of irritation in the region which drains into the lymphatic gland affected. Many instances might be taken to illustrate this. A child suffers from a conjunctivitis or corneal ulcer which gets well; perhaps at the time, possibly not till some time after, a gland is noted to be enlarged in the parotid region, and eventually suppuration takes place. Dentition is a common exciting cause. A child a year or more of age is cutting its molar teeth in the lower jaw; there is perhaps some tenderness or possibly ulceration of the edges of the gum; as the local tenderness subsides, one or two of the submaxillary glands are noted to be enlarged; they remain perhaps for months enlarged and hard, and then gradually soften down; the primary irritation passing away and being forgotten, it may be difficult, in the absence of a history, to say what was the exciting cause of the glandular enlargement. Lesions of the mucous membrane of the mouth, fauces, and nose are the most frequent exciting causes of glandular enlargements in the neck.

Besides dentition, attacks of scarlet fever or measles very frequently are followed by glandular enlargement, a result no doubt due to the tonsillar enlargement and catarrh of the throat and nose so frequently accompanying these diseases. Carious teeth, ulcerative stomatitis, cracked lips, are fertile causes of glandular enlargement. It must constantly be borne in mind that there is no necessary connection between the extent and severity of the primary irritation and the amount of glandular enlargement; the former may be slight and insignificant while gland after gland may become affected.

The exciting cause may be an injury to the skin by a wound of some kind, or the gland itself may be bruised or injured. The latter is not uncommon. A child receives a blow on the neck from a stone or a stick, the contusion perhaps quickly disappears, but some weeks or months afterwards a lump which proves to be enlarged glands is discovered.

An injury is perhaps the commonest exciting cause of the axillary and inguinal glands becoming enlarged. A chronic sore on the fingers or foot may be followed by caseous glands or cold abscesses, from the involvement of the lymphatics which drain the affected part. It is, however, far less common for the axillary and inguinal glands to be affected than for the cervical. Broken chilblains, which are so common in the strumous, may be the exciting cause of glandular enlargement.

Vaccination does in some cases appear to act as an exciting cause. An infant has a constitutional predisposition to caseation, or, to put it plainly, tuberculosis of irritated parts, and the irritation caused by the vesicles or post-vesicular ulceration may be the starting-point of strumous glands. Shortly after the "arm has taken" (or perhaps some weeks may elapse), some of the superficial cervical glands on the same side may be noticed to be swollen, and perhaps other of the cervical glands, both superficial and deep, join in the tubercular process. It is curious to note in these cases that the axillary glands appear to escape caseation, though they may enlarge; it is important to bear in mind that the cervical glands in the lower part of the neck have free communication with the axillary glands; moreover, they are joined by the lymphatics which drain the skin over the deltoid. By means of this communication irritative matters absorbed from the arm may give rise to inflammation and caseation of the cervical glands. In my experience strumous glands occur more commonly as a sequence of vaccination when calf-lymph rather than when humanized lymph has been used. This is probably due to the fact that calf-lymph is apt to set up more irritation than human lymph. I do not think there is the slightest evidence to show that any specific tuberculous material has been introduced into the system by vaccination, but the latter, like a simple wound or patch of eczema, has acted as the exciting cause only: the glands have become enlarged from the irritative matters passing through them, and, the child being predisposed to tuberculosis or strumous inflammations, caseation and slow suppuration have followed.

An important matter, in examining an enlarged gland for the first time, is to ascertain from what situation the irritative particles have been received. An examination must be made of the whole area drained by the affected gland. This, however, is not enough: irritative matters passing up the lymph-stream are by no means always arrested in the nearest gland, or at least do not always cause an inflammatory enlargement, or, if they do, the inflammatory trouble may subside without being followed by caseation. The infective particles may travel apparently by a by-route and affect glands in communication with those which drain the area in which the primary lesion is situated. Hence search should be made over a wide area for the source of irritation if nothing is found in the expected spot. It must also be constantly borne in mind that the original source of irritation may have disappeared long before the child comes under observation, and the friends may have forgotten it or overlooked it altogether.

The accompanying table may assist the memory when examining enlarged glands.

DISTRIBUTION OF THE LYMPHATIC GLANDS AND THEIR DRAINAGE-AREAS.[1]

GLANDS.	HEAD AND NECK.
Suboccipital *Mastoid*	drain posterior half of head.
Parotid	drain anterior half of head, orbits, nose, upper jaw, upper part of pharynx.
Submaxillary	drain the lower gums, lower part of face, and front of mouth and tongue.

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[1] From Curnow and Treves.

DISTRIBUTION OF THE LYMPHATIC GLANDS AND THEIR DRAINAGE-AREAS (*Continued*).

GLANDS.	HEAD AND NECK.
Suprahyoid	drain anterior part of tongue, chin, and lower lip.
Superficial cervical (lying beneath platysma)	drain external ear, side of head, and neck and face.
Retropharyngeal	drain nasal fossæ and pharynx (upper part).
Deep cervical:	
Upper set along carotid sheath	drain mouth, tonsils, palate, lower part of pharynx, larynx, posterior part of tongue, nasal fossæ, parotid and submaxillary glands, interior of skull, and deep parts of head and neck.
Lower set in supraclavicular fossæ	drain upper set of lymph-glands, lower part of neck, and join axillary and mediastinal glands.
	UPPER EXTREMITY.
Supracondyloid	drain three inner fingers.
Axillary	drain upper extremity, dorsal and scapular regions, front and sides of trunk and breast.
	LOWER EXTREMITY.
Anterior tibial and popliteal	drain the deep lymphatics of the leg, and receive some vessels from the skin of the leg and foot.
Inguinal:	
Femoral set (superficial)	drain superficial vessels of lower limb and partly of buttock and genitals, also perineum.
Horizontal set (superficial)	drain abdomen below umbilicus, buttock, genitals. The deep vessels of the lower limb go to the deep glands along the femoral vein.
Iliac	drain the pelvic viscera and the deep vessels of the genitals partly.
Lumbar	drain all the lower glands, uterus, testes, ovaries, kidneys.
Sacral	drain the rectum. Roughly, the umbilicus is the water-shed draining to the axilla and groin, but the vessels cross and overlap both vertically and horizontally.

The glandular enlargement in most cases is very insidious, is quite painless, and is free from any local tenderness. The enlarged glands are mostly discovered by accident, and may reach a considerable size before they are discovered. In acute disease, such as scarlet fever or measles, the cervical glands may become enlarged and tender during the course of the fever, remaining enlarged and indurated during convalescence and perhaps for many months or even years after. The size and situation of the glandular tumors in the neck necessarily vary: a single gland only may be affected; much more often several glands in close proximity are enlarged, or possibly the superficial glands with the more deeply seated glands near the same are affected.

The enlargement is essentially chronic; and the glandular tumor may remain for months, readily seen and felt, but giving the child no inconvenience, and without the slightest pain or tenderness. The progress of such glands is uncertain. In the first place, it is possible they may gradually disappear, possibly without caseating, or they may caseate without the caseous material softening into actual pus; in either case the tumor eventually subsides without any abscess being formed. The older the child and the better health it enjoys, the more likelihood is there that a chronic glandular tumor will eventually disappear. That caseating glands may eventually contract and become cretaceous there is abundant post-mortem evidence to show, as regards the cervical, bronchial, and mesenteric glands; we are driven to the conclusion that in these cases the system is able to resist the effects of the tubercular organisms and is not in a condition to favor pus-formation. On the other hand, it may happen that glands which have been much enlarged slowly diminish in size, then once more become active and go on to suppuration. On the whole, it must be said that after puberty the tendency to suppuration is much less than in early childhood, and, if a glandular tumor has existed for some time when puberty is reached, there is a good chance that it may gradually contract and disappear.

There is little doubt that in the majority of instances enlarged glands in scrofulous children end in suppuration. It is a common belief that suppuration is more common in the superficially-placed glands than in the deep, those glands beneath the deep fascia and sterno-mastoid suffering less than the superficial cervical glands. The bronchial glands and mesenteric glands also appear to suppurate less often than the external glands. In connection with this comparative frequency of suppuration, we must remember that the more deeply placed the glands are the more carefully protected are they from injury, for there is little doubt that a blow or other injury is frequently the starting-point of suppuration. Then as regards the bronchial and the mesenteric, they do unquestionably suppurate at times and discharge their contents by opening into a bronchus, the œsophagus, or intestine, possibly without in many cases this being detected during life. Certainly cretaceous glands may be found post mortem adherent to the bronchi and intestine, and, moreover, it is by no means uncommon to find post mortem a bronchial gland in the act of softening communicating by a fistulous opening into a bronchus. A caseous mesenteric or bronchial gland is far more likely, on account of its position, to start a tuberculosis, and so bring the end before suppuration occurs.

Suppuration.—How long may a gland remain enlarged without suppurating? No answer can be given to this question. We know that in weakly, scrofulous children an enlarged gland quickly caseates, and in such children the tendency to pus-formation is very great; not only so, but there is a marked tendency for the neighboring glands to become affected. But a gland may remain in a quiescent state or slowly go on caseating for many months or years, and the period during which it may remain passive is too variable to admit of any statement as to time.

When first enlargement occurs the glands are usually more or less elastic to the feel; later there is usually a more dense feel or a stony hardness, due to some extent to the fibroid changes which are in progress along with the caseation; probably also they are no longer movable, on account of adhesions being contracted between the capsule and the surrounding parts. When softening is in progress, parts of the tumor have a soft, fluctuating feel, and, as the pus accumulates, the tumor becomes more prominent and the fluctuation more decided. The skin sooner or later becomes undermined and thinned and of reddish tint; it finally gives way if not incised, and pus, usually thin and containing small cheesy fragments of broken-down glands, escapes. In many cases the thin, purplish skin over the gland sloughs, and an unhealthy-looking ulcer is formed, the edges being formed of overhanging infiltrated skin. It rarely happens that at the time the skin gives way the whole gland has softened down; in the majority of cases only a part of the gland has broken down at this time, and the caseous mass left inside prevents the healing up of the wound. Or perhaps the opening ceases to discharge, and scabs over for some days, then bursts open again, discharging thin pus and perhaps some caseous particles. In some cases the glands beneath the deep fascia may soften and discharge their contents through a small hole in the fascia, a long sinuous sinus being formed which is very slow to heal.

The scars left after the healing of the sinuses and abscesses depend upon the extent and chronicity of the suppurating process. The skin in the chronic cases may be infiltrated and contract; it may become adherent to the deep parts, so that the skin is puckered and drawn in. Ridges or inequalities or corrugations of the skin may be left when there have been ulcerations which have very slowly healed. The scar left after a quickly-healing incision is trivial, while the irregular cicatrices left after extensive suppuration and a very chronic course are certain to cause permanent disfigurement.

In many cases the pus formed is rather outside the capsule of the gland, the abscess being in this case *periglandular.* These abscesses are more common around the externally-placed glands than near the more deeply placed ones. An abscess of this kind, when its cavity is distended with pus, looks more like the cold abscess already described than like an abscess forming in a gland: when the abscess is opened, a caseous gland may frequently be found at its floor.

The symptoms present when the bronchial and mesenteric glands suppurate need not be given here in detail. In the case of the former the symptoms are often extremely indefinite, though occasionally an abscess thus formed makes its way to the surface by the side of the sternum or burrows down to near the ensiform cartilage. When a mesenteric gland forms into an abscess, the pus may find its way into the bowel or to the surface at the umbilicus; in some cases it may do both, and an intestinal fistula result.

Diagnosis.—In chronic glandular enlargement, if Hodgkin's disease can be certainly excluded, there is little difficulty in the diagnosis. The diagnosis between the glandular enlargements of Hodgkin's disease and scrofula is often impossible in the early stages. In the former the glandular tumor frequently varies in size, some weeks being large, apparently from being much congested, at other times being much less. There are often enlargement of the spleen, anæmia, and attacks of intermittent pyrexia; moreover, no suppuration occurs. The discovery of other signs of scrofula and a history of tuberculosis in the family would materially assist the diagnosis.

Treatment.—The preventive treatment of scrofula consists in placing the patient under the best possible hygienic conditions of life. These necessarily include protection from cold and damp, plenty of sunlight, well-ventilated apartments, good and suitable food, and a life largely spent in the open air. It is needless to say that it is impossible for us to place the majority of our patients under these favored conditions. It is the children of the courts and slums of our large towns who suffer most, and it is only when they are suffering from some aggravated form of scrofula that we are able to send them away (and not always then) to some sea-side or country sanatorium, where the conditions of life are favorable to recovery.

There can be little doubt, however, that scrofula in its worst manifestations is less common in England now than it was during the first half of the present century, or during the past times when multitudes of scrofulous folk used to crowd to royalty to receive the touch of the king's hand. Bad as are now the homes of the poor and hard as are their lives, a vast improvement has taken place, and a far larger proportion of the working-classes than formerly are well housed and well fed and have a fair knowledge of how to preserve their health. That there is less scrofula now than formerly is doubtless due to the facts that there is less tuberculosis than there used to be, and that the conditions under which children are brought up, whether in workhouse schools or in their homes, are less favorable to the development of scrofula.

The *general treatment* of those suffering from scrofulous manifestations consists in providing for them fresh, pure air, preferably residence at the sea-side, and a generous, well-regulated diet. If it is possible, children who so suffer should live or go to school at the sea-side, or at any rate spend the greater part of their time, both summer and winter, away from the smoke and vitiated atmosphere of our large towns. That scrofulous children are found both at the sea-side and in the country is certainly true; but in the majority of these cases it will be found that they are cases of hereditary disease, and, moreover, they may be subject to bad hygienic conditions as well at the sea-side or in the country as in towns. Certainly the children who mostly benefit by residence at the sea-side are those who have been brought up in towns or who suffer from the acquired form of the disease. It may be doubted if there is any specific influence exerted by the sea-breezes or the emanations from sea-water or sea-weed, as is often asserted: residence by the sea in most cases means more exercise, more fresh air, more sunlight, and better spirits than are enjoyed at home, and an improvement to the general health is the result, which reacts on the scrofulous disease. Chronically-enlarged glands and tuberculosis generally are certainly the outcome of weak and impaired health, and a change to the country or sea-side, with its many attractions and fresh air, is almost certain to improve the appetite and digestion and the various powers of the body.

Concerning diet, a supply of fresh milk is the first consideration; though in some cases the curd of milk does not always readily digest, as shown by the white, pasty stools. In the large majority of cases, if the conditions as to exercise in the open air, etc., are favorable to the digestion, a pint and a half or two pints of milk is not too large a quantity for a growing boy or girl. Much stress has been laid by some writers on the necessity of providing large quantities of fatty foods for scrofulous children, such as cream or bacon-fat. We must be guided in this matter by the child's powers of digestion, and, if fat is not well digested or produces nausea, it can hardly be of service. A fair supply of animal food, in the shape of butcher's meat, poultry, or fish, is certainly necessary for scrofulous children.

Care must also be exercised in the matter of clothing. Warm knitted stockings and gloves, as well as thick boots, should be provided for the winter, in order to guard as much as possible against chilblains and taking cold. Flannel or woollen garments must be worn next to the skin.

The morning bath or rub-down, especially if salt water is used, is a great help in promoting the circulation as well as in exciting a free action of the skin.

Scrofulous children can rarely bear cold water in winter. It is a good plan for them to stand in warm water while they are rapidly sponged down with tepid or cold water. Sea-bathing, with proper precautions, may be advantageously indulged in.

Concerning drugs, it cannot be said that modern medicine has supplied us with any that are specific in action or act as antidotes in counteracting the tendency to chronic inflammation and tuberculosis of the glandular system. We know of none which will prevent the growth and development of the tubercular bacillus in the system. Our only hope lies in improving the general health, so as to render the system less prone to chronic suppurations and caseating processes. Of the drugs most useful for this purpose cod-liver oil, iodides with iron, lime with hypophosphites, arsenic, and phosphorus are most in use at the present time.

Cod-liver oil undoubtedly holds the first place. It is not easy to say how it acts, as neither the iodides and bromides which it contains nor its oily constituents would seem at first sight, when taken in ordinary doses, to be sufficient in quantity to produce the improvement in the general health usually attributed to it. Yet there is a strong consensus of opinion that most cases of scrofula are benefited by its administration. It certainly agrees well with the majority of children, and it is astonishing how quickly they overcome their first repugnance to it. It is perhaps hardly necessary to say that before it is given care should be taken to see that the child gets a suitable diet, that it is not eating to excess, that its digestive organs are in good order, and that the stools are natural and of a good color. The oil may be given in the form of a good emulsion flavored with almonds and combined with lime in some form, or with extract of malt, or by itself. The choice must depend upon which form the child will most readily take and digest. After meals is the best time, a teaspoonful to a tablespoonful three times daily being the ordinary dose.

The oil seems to be beneficial in all the stages of scrofula and at all ages; it is most useful in the early stages of glandular enlargements, when caseous degeneration is presumably commencing and when there are still hopes that resolution or cicatrization may take place. Cod-liver oil is usefully employed as a local application to impetigo of the face, combined with or followed by some mild mercurial application, such as ung. hydrarg. ox. flav. Cod-liver oil is of more value during the cold than during the hot months of the year; it is best omitted during the hottest months.

Iodine and the iodides have long enjoyed a reputation in the cure of scrofula, both as an external and an internal remedy. As an application to enlarged glands iodine has almost universally been used, and for this it is a well-known popular remedy. Iodine is frequently employed in the form of mineral waters, such as the waters of Kreuznach or Heilbronn on the continent of Europe and the Woodhall Spa in England. Mineral waters containing iodine, if mixed with some mineral water containing an excess of sulphate of sodium, are often of value in the treatment of scrofula, a small morning dose being given sufficient to keep the bowels in action and the liver acting freely. The iodide of iron in the form of syrup is a very favorite remedy. Iodides are apparently of the most value in the early stages, before suppuration or extensive caseation has commenced.

The hypophosphites and phosphates of lime and iron have also been much used in the early and late tubercular or caseating process. Their value has been very differently estimated. Personally I am more inclined to prescribe them during the later stages, when caseation and suppuration are in progress, in those cases where a general tonic seems required. I am inclined to doubt if they have any special virtue in the curative process.

Arsenic, phosphorus, and mercury have all had their advocates in the treatment of glandular enlargements. They are medicines to be tried when cod-liver oil and the iodides appear to be ineffective in diminishing chronic glandular enlargements. They are most likely to be useful in the early stages, before caseation and suppuration have commenced.

Sulphide of calcium has been used during the suppurating stage, and has been strongly advocated by Ringer. I have never seen any great results from its administration.

Local Measures.—Any local source of irritation in mouth, fauces, nose, conjunctiva, face, and scalp in a child disposed to glandular lesions requires the most careful attention. Gum-boils, ulcers of the mucous membrane of the mouth, enlarged tonsils, patches of eczema, otorrhœa, should be treated, and if possible not be allowed to drift into a chronic state. It is useless to treat enlarged glands if there is some source of irritation in their drainage-area. For recently-enlarged glands, only soothing applications should be used, and they should be carefully protected from injury or from being chafed by hat-strings or by anything round the neck. A silk handkerchief loosely tied round the neck is the best protection. In this stage they may be gently bathed by means of a sponge wrung out of hot sea-water twice a day, and when carefully dried some lead lotion or belladonna ointment may be applied, all rubbing or friction being avoided.

If no improvement follow these means, some iodine ointment, preferably the ung. plumbi iodidi, may be tried, gentle friction being used. At the same time the general measures already spoken of should be used, and a residence of some weeks or months at the sea-side should be recommended. Much patience on the part of the friends may be required in this stage. It may be impossible certainly to say if caseation has taken place, though if the glands have been enlarged for several months and are firm and hard there is a strong probability that they have degenerated. Yet unquestionably glands which have remained enlarged for many months or even years will gradually disappear without suppuration occurring. In any case where the glands have remained enlarged for some time, the question of excision may be entertained, or a policy of masterly activity must be persevered in. All intraglandular injections are to be condemned: the injection of carbolic acid, acetic acid, or tincture of iodine is very likely to set up much irritation, and the result is that the neighboring glands become enlarged and possibly caseous.

Excision is no doubt the best course to pursue in the case of glandular tumors which remain at a stand-still for many months and have resisted all forms of general and local treatment. Unfortunately, excision has only a limited application. It is useful only in those cases where the gland-tumor is movable and superficial. Large gland-tumors which have contracted adhesions to the surrounding parts, more especially to the structures beneath, cannot be removed safely: to attempt to separate a mass of glands fixed to the large veins and arteries of the neck is an operation attended with great risk. The scar left after a successful removal of caseous glands is insignificant as compared with those left after chronic suppuration and long sinuous sinuses have formed.

The softening down and suppuration of a gland or glands are accompanied by a more or less elastic feel; as the pus accumulates the tumor becomes more prominent and the sense of fluctuation more and more distinct. Pus may, however, be present, especially when the deeper glands are involved, without any marked fluctuation being present. As soon as fluctuation is detected, no time should be lost in letting out the pus. It is unwise to poultice, or to delay till the skin is reddened and in part destroyed by the pressure of the pus making its way to the surface. Such skin is very apt to slough and leave a far larger scar than a clean incision through healthy skin. In all these cases chloroform should be administered, so that a thorough examination of the tumor can be made; an incision, say, half an inch in length, or sufficiently large to be able to insert one of Volkmann's spoons, should be made and the pus evacuated. The abscess should be thoroughly emptied and any caseous débris removed by a small scoop. A drainage-tube or piece of india-rubber tissue may then be inserted, and the wound dressed with carbolic oil or iodoform powdered on.

If the glandular abscess is allowed to open itself, or a small incision with imperfect drainage is made, the pus is apt to burrow and undermine the skin, unhealthy granulations form at the base of the ulcer, the skin sloughs, and an unhealthy, very chronic ulcer is the result.

While this treatment is fairly satisfactory in those cases where but few glands are affected and these are superficial, disappointment often results from the fact that the glands beneath the deep cervical fascia may caseate and keep up a constant source of irritation and a chronic sinus remains. In these cases the scraping must be repeated, the child being put under chloroform: all the unhealthy granulations must be scraped away, and the spoon pushed, if possible, along the sinus which passes through the deep fascia to the caseating deep glands and as much removed as possible. Thoroughly efficient drainage must be established.

Other methods of opening glandular abscesses, such as puncturing with the galvano-cautery (Treves), have been recommended and practised by various authors: the relative value of these and the details of procedure need not be entered into here; personally I must express my preference for incision or puncturing with the scalpel, and in chronic cases scraping away all softened materials by Volkmann's spoon, while at the same time ample drainage is provided.

Every one who has had much experience of the treatment of scrofulous glands must know how disappointing their treatment often is. An operation is undertaken, the broken-down glandular débris is removed, but some enlarged and probably caseous glands are discovered beneath the deep fascia, with or without a sinus leading down to them; excision of these may appear to involve some risk on account of their deep connections; in such cases it is probably best to wait, in the hope that they may gradually disappear. But mostly a further trial of the patience of the friends and medical attendant is pending, inasmuch as the operation-wound heals up only to break out again and again, and another operation some months or years later has to be undertaken.

Prognosis.—In most cases of glandular enlargement a cautious prognosis must be given, and it is unwise to promise that an operation will certainly bring the glandular troubles to an end.

TUBERCULOSIS.

By A. JACOBI, M.D.

THE definition of the term "*tubercle*" has experienced a great many changes. Originally it means a prominence or protuberance. In the Latin translations of Hippocrates it stands for cold (caseous) abscesses. Francis de le Boe (Sylvius, 1614–1672, in "Praxeos Medicæ Idea Nova," 1667–1674) applies the name to small bodies met with in different tissues and developed from presumed invisible glands, Baillie (1761–1823) to an abnormal product of scrofulous origin, Bayle (1774–1816) to an independent specific neoplasm endowed with great tendency to caseous degeneration. With him, indeed, the latter was characteristic of, and solely found in, tubercle. He and Laennec (1781–1826) looked upon the tubercle as the cause of consumption (phthisis), the latter author adding to pathology and nomenclature the term "tubercular infiltration." Lebert (1813–1878) described the microscopical "tubercle corpuscle" as consisting of disintegrated cells, or free nuclei, thus enabling everybody to discover tubercle wherever it did and did not exist. Schönlein (1796–1848) was the first to use the term "tuberculosis."

According to Virchow, the tubercle is an organized, though not vascularized, neoplasm composed of round cells with very vulnerable and deciduous membranes and very numerous nuclei. These may be so copious, indeed, that the membranes are sometimes not discovered. The tubercle is small; even the smallest, however, is often a conglomerate; it is of gray color, turning yellow through caseous (fatty) degeneration, which begins in the centre. It leads to tubercular "infiltration" by the aggregation of many tubercles and secondary inflammation in the neighborhood; or to ulceration; or to the hardening of the small body ("fibrous tubercle") by disintegration and absorption of the cells and the increase of the, originally, scarce and thin connective tissue.

The small epithelioid cells with their nuclei were soon found not to be the only microscopical constituents of the tubercle. Virchow, Rokitansky, and many others, found "giant cells," and Th. Langhans claimed them as almost constant constituents. They are of spherical shape, contain from twenty to a hundred nuclei, with leucocytes in their periphery, and a very fine reticulated tissue between these constituents.

The reticulated tissue, and giant cells, are mostly found in chronic tuberculosis. In this process a considerable amount of fibrillar connective tissue is met with in the periphery of the deposits. In the acute process small spherical cells are more frequently found; they are also copious in the periphery of tubercles when they undergo caseous metamorphosis. This latter process is apt to spread into the surrounding congested or inflamed tissue; quite often the very caseous masses contain tubercles still intact.

To identify, however, caseous degeneration with tuberculosis would be a mistake. The former is no neoplasm, nor intimately connected with a specific neoplasm, but a retrograde metamorphosis. It is not characteristic of any single pathological tissue or condition, for, besides being found in tubercle and inflammatory deposits, it may be the final stage of development in pus, cancer, and typhoid infiltrations.

Nor are giant cells pathognomonic of tuberculosis. They are found in the disintegrating osseous substance, in the cavity of the uterine sinuses near the insertion of the placenta, near foreign substances experimentally introduced into the peritoneal cavity, in pneumonia, syphilitic endarteritis and gummata, in healthy granulations, sarcoma, and actinomyces, and in the subcutaneous tissue of animals into which silk, hair, and other foreign bodies had been introduced for the purposes of experimental research (Birch-Hirschfeld).

Thus, neither the histological structure of the tubercle nor its tendency to caseous degeneration suffices to characterize tuberculosis as a specific disease of an infectious nature. The latter has long been assumed to exist by common consent, and appears to be finally demonstrated by R. Koch's discovery of a specific bacillus which gives rise to a local irritation and the formation of the specific noduli. Modern pathologists have agreed in this, that only such products, though histologically the same or similar, as contain, and result from, the specific bacilli, deserve the name of tuberculosis. Thus, tuberculosis is defined as an infectious disease which shows, as the result of the immigration and proliferation of a specific bacillus, conglomerates, small or large, consisting of cells with few or many nuclei and nucleoli, and (as they are without blood-vessels) disposed to undergo speedy caseous degeneration. In the latter condition, when recent, the tubercle is called yellow. The accumulation of a great many yellow tubercles forms what is called an infiltration. Calcification is the result of copious hyperplasia of cellular tissue round a tubercular infiltration. Softening is a more frequent occurrence, and leads to the disintegration of viscera, cold abscesses in the subcutaneous tissue, and alterations of mucous membranes.[1]

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[1] Thus, according to the present state of the pathological doctrine, tuberculosis demands the presence of the bacillus. Still, there are processes which are tuberculous in everything but the bacillus. Thus, Malassez and Vignal found zooglœa only, mostly without bacilli, in "tubercles" produced in experimental proceedings. Similar results were obtained by Cartro and Soffia: their zooglœa could be inoculated successfully. Biedert reports the case of an acute pulmonary tuberculosis without bacilli. Ribbert met with small bodies consisting of

Etiology.—*Congenital predisposition* need not be identical with *hereditary transmission.* The former may result where numerous children are born of non-tuberculous parents in too rapid succession; from puny development of the infant; from under-size of the heart, from anæmia based upon stenosis of the pulmonary artery, or from congenital shortness or premature ossification of the costal cartilages in the upper part of the chest, by which the apices are prevented from expanding and the circulation of the blood is impeded.

Hereditary transmission of tuberculosis has been claimed as a fact by common consent, because of the frequent occurrence of the disease at an early age, and the great number of cases observed in a family. Vogel looks upon heredity as the principal etiological factor. For he observed that a child of a healthy family when living with a predisposed family under the worst possible hygienic surroundings would not suffer, while all the rest would succumb. Thus he concludes that external influences are injurious to those only who are predisposed, no matter whether heredity is visible in the propagation of either a predisposition or a virus. It is the latter in which Baumgarten believes. Brehmer, however, thinks but little of either mode of transmission, because "not more than one-third or one-half of all the cases" occur in families in which there is a multiplicity of cases.

Hereditary transmission ought not to be presumed to exist at all except in cases which occur at a very early period of life. Infants of tuberculous parents, though they fall sick with tuberculosis, or atrophy, or marasmus, when but a few months old, *may* suffer from the consequences of a germinative process, but their disease *may* also be due to direct contagion, or tuberculous food. Still less conclusive are those cases which make their appearance in bones or glands after a number of years only. It is mainly this class of cases that has given rise to the theories based on predisposition, or on the gradual transmutation of scrofulosis into tuberculosis.

Hereditary transmission of tuberculosis is not accepted by a number of the most critical pathologists. Benda denies the possibility of the transmission of bacilli through sperma which has its origin in nuclei not infected by parasites. He did not find them in sperma secreted by tuberculous testicles, nor in that of phthisical patients whose testicles were healthy. Virchow takes it for granted that tuberculosis resulting from infected sperma ought to develop at a very early period of life, in which it is rare, or at birth, when he knows of no such case. He even found the fœtus without tubercles when the mother had tubercular endometritis, and does not admit the possibility of a direct transmission unless the circulation

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lymphoid and other cells, giant cells included, without bacilli, which he prefers to call multiple lymphomata solely because of the absence of the micro-organisms. Eberth describes the same condition under the head of "pseudo-tuberculosis." Biedert (Lehrb. d. Kinderkr., 1887, p. 532) suggests that there must be either an affection which cannot be distinguished from tuberculosis, or a condition of the bacillus which renders its recognition impossible.

of the placenta be abnormal. Still, under certain circumstances the blood-vessels of the placenta are known to be pervious. Coloring substances have been found to penetrate into the body of the fœtus by Reitz and Mars, while other experimenters have but negative results. The bacilli of anthrax have been found in the fœtus by a single observer, those of septicæmia by a very few. That, however, some medicinal substances will traverse the placental circulation and be found in the fœtus, we know; also that syphilis, variola, relapsing fever, malaria, may be transmitted from the mother to the fœtus. Such facts exist, though they may be explainable only by the assumption of a morbid alteration in the walls of the blood-vessels of the placenta or its insertion.

There are, however, some facts which render the theory of a direct transmission of tuberculosis somewhat probable. Thus, in the spermatic canals of non-tuberculous testicles, in eight men dying of phthisis, C. Jani found bacilli five times, and four times in the prostate glands, out of six autopsies. Besides, there are a few cases of congenital tuberculosis of animals on record. I willingly exclude Czokor's calf of three weeks, and the two calves of Hertwig's of two and four months; for all of them may have contracted acute tuberculosis after birth by direct communication or the milk sucked from a diseased udder. But Johne has the report of an eight months' fœtus of a calf, with universal tuberculosis.

In the human race no case of a similar nature has been known, but in 1861 I attended a phthisical woman in her first confinement. She belonged to a consumptive family, had suffered herself before she got married, and died in the third week after confinement. The fœtus was born at the end of the seventh month of utero-gestation, and lived a few minutes only. There were numerous gray miliary tubercles in the tissue of the liver near the surface, a few in its peritoneal covering and the spleen, and on the pulmonary pleura. The father was healthy and remained so for years. Thus this isolated case, the only one of the kind ever observed by me, appears to prove the possibility of a direct hereditary transmission from the mother to the offspring. Epstein's two hundred babies of tubercular mothers yielded a negative result. There was but one of them who had tuberculosis at the age of ten weeks.

There are other observations which appear to prove that hereditary transmission is more frequent than is allowed by those who insist upon inhalation as the only cause of tuberculosis. Indeed, such observations are numerous. In the earliest period of life, tuberculosis is mostly found in the lymph-bodies and the bones. Why not first in the lungs, if inhalation brought it on? It has also been noticed that healthy babies, raised in tubercular families, are not liable to be infected, while the children of parents who died of tuberculosis while the former were quite young, would still die of tuberculosis, though removed to healthier quarters.

Though the cases of tuberculosis in the very first weeks of life be ever so scarce, we cannot say that any *age* is entirely exempt. Baumgarten met with cases of tuberculosis at the age of one month which were so advanced as to make its starting during fœtal life probable. Steiner and Neureutter report cases of tuberculosis occurring at the age of eight weeks, F. Weber cavities at less than three months, Demme on the twelfth day, Steffen at three weeks. Demme has another case of a baby three weeks old with tuberculosis of the intestine, and bacilli; and another one of four weeks with pulmonary cavities. Between the fourth and sixth months of life I have met with it in a number of instances. Lorey gives the ages of one hundred and sixty-two tubercular cases among children as follows: from the first to the third month, one; from the third to the ninth, eleven; from the ninth to the twelfth, thirty-one; between the first and the second year, fifty-five; from the second to the fourth, forty-one; and from the fourth to the twelfth, twenty-three. In Biedert's tables containing the ages of the young affected with pulmonary tuberculosis six and eight-tenths per cent. were observed under one year of age, forty-eight from the first to the fifth, twenty-seven from the fifth to the tenth, and eighteen per cent. from the tenth to the fourteenth year. Thus tuberculosis is comparatively rare under one year, undoubtedly because of the comparatively few opportunities for infection: as a rule, these early cases are due to, or connected with, the existence of catarrhal pneumonia, or intestinal difficulties, or marked scrofulous disposition. Between the ages of two and four years it is quite frequent, the lungs, pia mater, and intestine being the very organs through which it is apt to become fatal. In the former two, in early childhood it is not readily of a primary character; at that age the intestines, bones, and lymph-bodies are more liable to be the seats of the original inlet than the lungs. These are more easily affected, primarily, in advanced childhood, and about the period of puberty.

The former belief that acute tuberculosis was more frequent in the young, and the chronic variety in the old, holds good no longer, since a large number of diseases of the bones and lymphatic glands have been recognized to be of a strictly tubercular character. It is particularly the latter organs that are exposed to infection, because of their superficial location, and, in infancy and childhood, the comparatively large size of the lymph-ducts, the greater vulnerability of the surface which facilitates the access of a virus, and the physiological activity of the whole lymph-circulation.

This is but one of the many instances of the peculiarities of disposition depending on the nature of the tissues. Others are found in the different degrees of the energy of respiratory movements, the various conditions of the epithelium, the secretion of the muciparous glands, and the circulation in the lungs. In the latter, tuberculosis is not so frequent in the apices of the young as in those of the adult, because of the larger amount of air entering them in the former. In them, indeed, it is the lower parts of the lungs which are often the preferred seat of the malady. And those lungs which are anæmic, either on the basis of general anæmia or as the result of the stenosis of the pulmonary artery, are much more liable than those affected with chronic venous stasis depending on emphysema, kyphosis, or congenital or acquired disease of the heart.

Animals have been made tubercular by the *inhalation* of tubercular sputum. The viability of the bacilli and their spores is such as to render them dangerous though, or because, they have been in a dry state on the floor of the room, in carpets, linen, or clothing, for a long period. They will not easily locate in the external parts of the respiratory organs where the air is cool and its current capable of carrying them out as well as in. That "bad" air is a cause of general tuberculosis has always been accepted as undeniable. The latter would increase with crowding. In the foundling asylum of Stockholm, Abelin noticed that the proportion of cases of tuberculosis would increase with the number of inmates. In the light of modern pathology the "bad" condition of the air may signify as well the prevalence of bacilli as the presence of injurious gases and the diminution of individual air-space.

Inhalation has always been considered as one of the principal sources, or the principal source, of acquired tuberculosis. Many of the reports, however, which were meant to prove the frequent occurrence of such cases, leave ample room for doubt: thus, for instance, those of the ten new-born babies said by H. Reich[1] to have been infected by a consumptive midwife, who had the unfortunate habit of insufflating the respiratory organs of the young with her own breath.

In order that virus, or a bacillus, may find a resting-place, the surface must be in a morbid condition. A mucous membrane of normal consistency and function is not very liable to admit infectious diseases. Neither diphtheria nor tuberculosis finds a safe nest on a healthy membrane. As long as a mucous membrane is covered with normal mucus and protected by vibrating epithelium, foreign bodies, from particles of carbon and metal to bacilli, are liable to be expectorated. Only the air-cells which have no fimbriated epithelia allow bacilli to rest and to develop. All the other surfaces of the respiratory organs are endowed with means of self-defence. The latter, however, is greatly interfered with either by an abnormal structure of the integuments or by actual lesions. The former may be inherited from parents suffering from chronic infectious diseases, such as tuberculosis or carcinosis, or acquired by previous exhausting ailments, anæmia, or chlorosis; the latter may result from measles, whooping-cough, typhoid fever, or scarlatina, or inflammation or gangrene of the lungs, which thus give rise to a predisposition to tuberculosis by having prepared the surface for the admission of the virus.

The bacillus, however, is not found floating in the air and ready for inhalation unless under exceptional circumstances. To be inhaled it must be dry. As long as sputum is moist, or, after having been dry, is again exposed to moisture, it cannot be mixed with the air and thus enter the lungs of another person. Besides, the bacillus has a greater specific gravity than air, and falls to the ground. But it may adhere to bedclothing, or the bedstead, or the walls of the room, or the floor which has been soiled. Thus, the children of a phthisical mother may all be infected by their close contact with her and her surroundings, while a nurse, or the husband who goes about his business, is not suffering. Thus, also, the phthisical patients in the wards of a hospital are uninjurious as long as no expectoration is permitted anywhere but in a spittoon containing some water.

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[1] Berl. Klin. Woch., 1878, No. 37.

Still, the frequency of tuberculosis makes its transmission easier than the explanation of the latter in every case. Thus, for instance, Spillmann and Haushalter,[1] having made the observation that flies would concentrate round the sputa of tubercular patients, kept a number of them under a bell-glass, where they died the following day. Their excrements deposited on the glass and the contents of their abdomens exhibited an abundance of bacilli tuberculosis. As these bacilli are very hardy, their transportation by the fly to the food of human beings, and those contained in the dried remains of the fly, appear to open a possibility to the transmission of tuberculosis to an almost incredible degree.

Besides, the bacillus of tuberculosis is of slow growth, and thus facilitates self-protection on the part of the endangered organ and organism; though, on the other hand, it is very tenacious of life. For a five-per-cent. solution of carbolic acid destroys it after twenty-four hours only, and a still longer time is required by a one-per-mille solution of bichloride of mercury. It does not even perish when exposed to a high degree of heat: G. Cornet exposed mattresses to the effect of public steam-heating apparatuses six times, and still found bacilli uninjured and active.

The entrance of tuberculosis through the *skin*, or *wounds*, is among the possibilities. As long, however, as the skin remains in a normal condition, it affords protection against the entrance of tuberculosis. But abrasions and wounds create a disposition. Still, the development of bacilli appears to require a higher temperature than that of the very surface, and a sufficient time for their sure installation. Thus is explained why the number of authenticated cases of the invasion of tuberculosis through the skin is still limited. Willy Meyer collected[2] twenty-eight such cases; M. B. Schmidt and others have since published a few more. Eighteen of the twenty-eight were those of Jewish infants subjected to ritual circumcision, which permits, or requires, the sucking out of the wounds by the lips of the operator. The incubation-period lasted from ten to fourteen days; after that time the first symptoms showed themselves as inguinal adenitis. Of the eighteen, nine died, five exhibited symptoms of scrofula, and four were not under observation afterwards. In a few (adult) cases of wound-infection the disease remained local : still, it is probable that, as the development of tuberculosis is a gradual one, many isolated cases due to local infection may become generalized after a while. Chronic inflammations of the skin may frequently give access to the virus. Demme found chronic impetigo in four hundred and thirty-seven out of eight hundred and seventy-three cases of diseases of the bones and joints.

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[1] La France Méd., 1887, t. ii. No. 101.

[2] N. Y. Med. Presse, June, 1887.

In the Congress[1] assembled at Paris in July, 1888, for the study of tuberculosis, Dr. Degive, of Brussels, alluded to the possibility of transmitting the disease by *vaccination*. In his city the calf from which the virus has been taken is killed; when it is found to have been healthy, the virus is used for both human vaccination and the artificial infection of other animals. But even the danger from virus taken from a diseased animal is but very slight. For the bacillus does not easily penetrate through merely superficial wounds, and certainly not into the serum of the vesicle any more readily than is done by the syphilitic poison. Thus no danger appears possible unless blood be mixed with the serum of the vesicle used for the vaccination of the human being.

One of the inlets of tuberculosis is undoubtedly the *alimentary canal;* indeed, there are some who attribute every case—or almost every case—of tuberculosis in the young infant to the influence of food containing the bacillus. Koch has established the fact that the latter may pass through the stomach and remain intact; in the intestinal canal it may be found mixed with food and nasal and pharyngeal mucus. In the healthy digestive organs it will do no harm; indeed, the normal stomach will not permit it to live. But the absence of acids in the feverish stomach, and the changes produced in the mucous membrane by abnormal digestion, sedentary life, emotions, serious illness, or constitutional ill nutrition of the digesting surfaces, may yield conditions favorable to the invasion.

This may take place when the bacillus is an *accidental admixture* to the ingesta, or is swallowed with the *expectoration,* all or most of which is carried downward by infants and children. Thus a constant auto-infection is added to the original disease when this is located in the lungs. But the main opportunity for the invasion is furnished by the *meat* and *milk* of tuberculous animals. In the slaughtering-houses of Rouen there were 1.43 per mille tubercular heads of beef, 0.09 of calves, and 0.38 of hogs: these figures are the average of the four years between 1884 and July of 1888. There were furnished in Montauban, in the course of seven years, 4.07 per mille of tubercular beef among all that were slaughtered. Fürn found twenty-two tuberculous geese in three hundred and sixty-five autopsies, Reimann sixty-two hens among six hundred, and eleven pigeons affected with the same disease among one hundred and thirty-eight autopsies. Walter K. Sibley found the bacillus mostly in the peripherous parts of caseous masses removed from fowls,[1] and in undoubted lymphomata, undergoing central necrosis,[2] taken from a serpent, also from a peacock and an owl. Among sheep and goats, which move in fresh air, there were but few affected with tuberculosis. The influence of air and exercise is quite marked, so much, indeed, that T. Spillmann found from thirty to forty per cent. of all the stall cows of Nancy to be sick with tuberculosis. Even more than this percentage of tubercular animals is obtained by Brush for those which are "improved" by persistent breeding in. The opinions in regard to the danger attending the eating of meat taken from tuberculous animals are by no means uniform. In the muscular tissue the bacillus develops but incompletely: indeed, it has been observed to die within six days. E. Nocard found invariably that the inoculation of meat juice taken from tubercular animals had but little success; and Arloing, another of the great veterinarians of France, had the same results in his experiments. Both, however, found an abundance of bacilli in the glands, kidneys, spleen, and liver of the diseased animals. All of these organs are declared to be very dangerous under these circumstances, but the meat is deemed to be innocuous or but little dangerous in all but a very few cases. G. Butel, however, considers the meat of tubercular animals to be injurious under all circumstances. Baillet fears it only when the malady has rendered the animal thin and languid; but, again, Veyssière advises the exclusion of the meat of every animal suspected of tuberculosis, and emphasizes the fact that hogs are very subject to the disease.

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[1] Congrès pour l'Étude de la Tuberculose, Paris, 1889, p. 157.

The same difference of opinion prevails in reference to the milk of tuberculous animals. B. Bang found that milk of phthisical women could be inoculated with no danger at all. The inoculation of milk taken from twenty-one diseased cows yielded a trifling success in but two instances. But the majority of authors see more harm in such milks, and there are those who, like V. Galtier, find bacilli and danger not only in the milk of infected cows, but also in its products, such as cheese, buttermilk, and whey. Koubassoff attributes great danger to every milk of tubercular cows, Bollinger and Nocard only to that which is taken from tubercular udders. Still, authors of equally high reputation, such as Bouley and Bang, do not deem the presence of a tubercular mastitis necessary; the latter is declared to be a rare disease by Nocard, a frequent one by Degive and Van Hertsen. Upon this, however, all appear to be agreed, that heat destroys the dangerousness of milk obtained from infected animals. From 60° to 75° C. diminish it considerably. Milk heated to 85° C. is deemed safe.[3] For thirty years I have insisted upon the necessity of avoiding raw milk among the foods of children.

Localization.—There is hardly an organ in the infant or child which may not be affected by the tubercular process.

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[1] Trans. Path. Soc., London, 1888.

[2] Virch. Arch., vol. cxvi. p. 104, 1889.

[3] Congrès pour l'Étude de la Tuberculose, Paris, 1889.

Cutaneous tuberculosis may appear in a primary and secondary form.

The primary form, or lupus, is not very frequent during childhood, but still many of the cases met with in adolescence and advanced age date from early life. It has a very slow development. It is found on the face and extremities, and sometimes extends to the mucous membrane of the mouth, nose, pharynx, and larynx. On all of the latter it yields a diffuse infiltration, not nodulated, of gray color and irregular surface, interrupted by rhagades and ulcerations; while in the former it consists of red or brownish noduli, which are deeply embedded in the corium, with an occasional tendency to disintegrate and either form ulcerations or result in a desquamative process or a cicatricial atrophy. Anatomically, it is composed of small nests of round cells embedded in the interior of the corium, giant cells (mainly in the large noduli), and hyperplastic proliferations resembling those of epithelial carcinoma. It contains the tubercle-bacillus, and tuberculosis can be produced by its inoculation. It is not uncommon to find general tuberculosis in other members of the same family. Still, the tubercular nature of lupus has been doubted by Kaposi, because of the paucity of the bacilli in the morbid changes, the non-appearance of general tuberculosis in the same individual after a long duration of the lupus, the impossibility of multiplying lupus by inoculation, and the almost universal immunity from lupus of the other members of the same family.

The secondary form of cutaneous tuberculosis starts from tubercular joints, mucous membranes, and caseous and suppurating lymph-bodies. Fistula in ano may give rise to it, as, indeed, tuberculosis is apt to appear near the mouth, the anus, and the genital organs. In one of my cases, that of a girl of seven years, the process commenced from a neglected abscess in the right axilla. The fistulous and undermined ulcerations spread in every direction, extended over the chest, resulted in tuberculous abscesses extending towards the abdomen, and finally in pyothorax, with general miliary tuberculosis. This form is not nodulated, not hard, and not of that slow growth extending over years so characteristic of lupus, but is more ulcerous, of irregular outlines, and with but little infiltration. From syphilis of the cutis it is best diagnosticated by its very slow growth and the absence of the indurated boundary peculiar to the syphilitic ulceration.

In the *joints and bones* tuberculosis is frequent. Many of the cases of caries are of that nature; a large percentage of the cases of ostitis of the foot and ankle and of spondylitis belong to this class; also a number of cases of caries of the mastoid process, with or without facial paralysis, and of otitis media, extending to the bone. The fungous arthritis is pre-eminently tubercular, for bacilli may be found in many a case. This class of cases is quite dangerous when left alone to such an extent as to lose its local character. If removed by an operative procedure, the localized tuberculosis loses its dangerous nature, and general infection may be avoided.

On the *pleura*, also, tuberculosis may be either primary or secondary. In infancy and childhood the former occurrence is but rare; as a rule, tubercular pleurisy, or tubercles on the pleura, are met with in generalized tuberculosis. In that case the tubercles are small or large, gray, yellow, or caseous; large caseous tubercles are mostly found on the point of contact of the adhering pleura. The assumption that every pleurisy is tubercular is based on theory only; for the cases of chronic pleurisy, of thickened pleura carried many years without a trace of tuberculosis, are by no means rare. The fluid of the pleural cavity found in tubercular pleurisy is either serous or purulent; in very rare cases there is blood mixed with the serum, or clear blood. Tuberculosis of the *pericardium* I never found, except complicated with that of the pleura, or as a part of general acute miliary tuberculosis.

The low temperature of a part of the *nose*, the constant motion of the air-current, and the presence of secretion on the mucous membrane render primary tuberculosis of that organ a rare occurrence. Still, the so-called scrofulous ozæna is very often tuberculosis; even that, however, is quite often not primary, but the result or accompaniment of neighboring or general tuberculosis. In and about it, giant cells and bacilli are met with. The majority of cases of nasal tuberculosis are of a secondary nature. It is either miliary, the nodules are gray or yellow and disintegrate very readily, or it exhibits large ulcerations of irregular shape, or, thirdly, large tumors, mostly on septum or conchæ; they rarely extend to the bone, and consist of connective and granulation tissue and miliary tubercles.

Both primary and secondary tuberculosis of the *pharynx* is relatively scarce in infancy and childhood, though its surface be constantly exposed to the contact with infected expectoration. Still, I have seen quite a number of cases, mainly between the ages of seven and fourteen, in which both miliary tubercles and painful tubercular ulcerations were found on the soft palate, tonsils, posterior wall of the pharynx, and nares. In a few cases the ulcerations were so deep, and the accompanying œdema so extensive, that fluids would escape through the nose. In one case the diagnosis from syphilis could not be made except after a certain time; as a rule, however, syphilitic ulcerations are less numerous, but deeper and steeper, and apt to heal under specific treatment.

Tuberculosis of the *larynx* is not so frequent in children as in adults. Of primary cases, or such as I could take for primary, I have seen but very few. At all events, when the diagnosis of tuberculosis of the larynx had been made, the appearance of pulmonary symptoms was but a question of a short time. Still, there is no reason why bacilli should not locate in the mucous membrane predisposed by the presence of catarrhal erosions, mainly on the vocal cords and in the interarytenoid space, also on the edges and the inferior aspect of the epiglottis. It is on these localities that both miliary tubercles and ulcerations are sometimes found. Mild symptoms of catarrh, hoarseness, cough, are observed at an early period, speaking and pressure are painful, the expectoration contains pus, blood, bacilli, and sometimes elastic fibres, and the laryngoscope reveals an incomplete closure of the glottis, the presence of tubercles or ulcerations, and, occasionally, localized œdema (perichondritis).

In the *thymus* gland tuberculosis is not rare at all. It was met with by Dr. Koplik and myself three times in sixty autopsies of infants under a year, twelve of whom had generalized tuberculosis. Sometimes it is found in the thymus, while no other organ, and no other member of the same family, is affected.[1]

Tuberculosis of the *peritoneum* is rarely a primary disease, and then acute or with high fever and urgent symptoms. It is mostly secondary, a part of general tuberculosis, or connected with protracted suppurations, or depending on embolism. It may originate in more advanced age in uterine tuberculosis, the tubes being the connecting link, or result, in the child, from intestinal ulcerations or disintegrated mesenteric glands. Sometimes it is quite local, in an intestinal adhesion opposite an open or cicatrized ulceration; in other cases it extends over large surfaces and may result in wide-spread adhesions, contractions, perforations, and hemorrhages. The tubercles found may be small or large, gray, yellow, or caseous. The accompanying inflammation may result in the effusion of large quantities of serum containing much albumen, or in fibrinous thickening of the peritoneum of the abdominal wall, liver, spleen, and omentum, with considerable glandular swelling, or the formation of large masses of exudation, between which and malignant tumors, mainly sarcomata, the diagnosis may be quite difficult. Still, not all of these exudation-tumors are of tubercular nature. I have seen them, from the size of a hazel-nut to that of a goose-egg, sometimes in large numbers, as the results of a chronic exudative peritonitis of non-infectious character, and diminishing in size and disappearing altogether until a permanent recovery. The temperature may not be very high ("peritoneal tuberculosis"), or may be quite elevated ("tubercular peritonitis"); other symptoms, such as fluctuation, pain, dulness on percussion, meteorism, diarrhœa or constipation, jaundice by compression of the ductus choledochus, obstruction by pressure on, or contraction of, intestines, depend on the extent of the affection and its more or less acute character. In the case of a boy of seven years who died with general tuberculosis, I found, beside large quantities of serum, which filled the abdominal cavity, complete adhesion and thickening of all the intestines, so as to yield the consistency and hardness of pasteboard. In very young children isolated peritoneal tuberculosis is but rare; it is, however, a frequent occurrence in generalized miliary tuberculosis; in older children I have seen many cases in which—mostly on the foundation of glandular degeneration—the disease, usually of a chronic character, appeared to have been the starting-point of the general affection.

The tuberculosis of the *liver*, *spleen*, and *supra-renal bodies* is, with very rare exceptions, secondary to, or a part of, general tuberculosis. Those organs are generally affected only towards the fatal termination, the tubercles being gray or yellow, seldom large and caseous.

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[1] Congrès pour l'Étude de la Tuberculose, Paris, 1889, p. 238; Transact. of the Assoc. of Amer. Phys., 1888.

The *kidneys*, both capsule and substance, participate in generalized tuberculosis. A large tubercle, of the size of a hazel-nut, I have seen in the left kidney of a girl of eight, who exhibited caseous degeneration of many of the bronchial and mesenteric glands, and cavities in both apices. Such a condition may be presumed to exist when a tuberculous child exhibits hæmaturia or dysuria. Tubercular ulcerations of the *ureters* or *bladder* I have not met with.

Tuberculosis of the *vulva*, in a girl of seven years, I have seen but once. It appeared in the shape of lupus complicated with angry-looking ulcerations, the edges of which were lined with miliary tubercles. The uterus and its appendages, except in cases of general miliary tuberculosis, I have not seen affected.

Tuberculosis of the *testicles* is not quite rare. Henoch has seen a few cases at the age of from one and a half to seven years, the epididymis being hard and nodulated, occasionally; and Koplik has but lately described the case of an infant. Sometimes it is primary, but almost in every case there was tuberculosis in other organs, mainly in the bones (caries) and peritoneum. My youngest case was seven months old; at that time the right testis was of the size of an egg, hard, and irregular. It had been known to swell but six weeks before it was presented. It grew rapidly to double its size, and had not lost its hardness when the infant died of general miliary tuberculosis (meningeal, pulmonary, and mesenteric, mainly) within a few months. In the case of a boy of three years, who also died of (chronic) general tuberculosis, the right testicle was of the size of a walnut when first seen, and did not increase much in size when caseous degeneration took place, and both testis and the adhering scrotum were pierced by a number of suppurating fistulæ. Cicatrization of such fistulæ has been observed, but none of my few cases lived long enough for such a termination of the local process.

The interior of the *intestinal tract* may become the seat of tuberculosis through the medium of the circulation, or by the ingestion of bacilli contained in sputum, meat, or milk. I know of no instance where intestinal tuberculosis, well developed, was proved to be the primary or sole affection, nor is it probable that tuberculous processes should develop to any extent without implicating the neighboring glands at least; but it must be admitted that there may be such a possibility. The solitary follicles and Peyer's patches are the main localities for tubercular deposits; their forms are those of miliary nodules or infiltrations, their changes the same as those which take place in other organs. They disintegrate in the centre, ulcerate until they perforate, unless peritonitic adhesions prevent this ominous termination, and give rise to secondary miliary deposits in and round their very edges. These ulcerations are found mostly from the lower part of the small intestines to the ascending colon, but also to the rectum. According to their seats, they produce pain, diarrhœa containing mucus and blood (in one case Biedert made the diagnosis by the presence of tubercle-bacilli in the evacuations), and tenesmus.

The lymphatic *glands* are involved in almost every tuberculous process. That swelled "scrofulous" glands preceded, or were complicated with, tuberculosis, was acknowledged to be a fact long before the bacillus was recognized. The lymph-bodies of the neck and omentum, and the bronchial and retroperitoneal glands, are among those most frequently affected. Their morbid condition remains sometimes latent for a long period. When they undergo caseous degeneration and suppuration, they may give rise, through embolism, to pyæmia or general tuberculosis, or, when near the surface, to tuberculous ulceration and fistulous destruction of the skin.

Their relation to tuberculosis has been described in the previous article on scrofulosis. Nothing could be said here that would add anything to Dr. Ashby's masterly and instructive exposition of the subject.

As far as the subject of the abdominal glands is concerned, we shall have to return to it in the discussion of tabes mesenterica. There the consideration of intestinal tuberculosis will again occupy our attention. The bronchial and tracheal glands in their connection with the tuberculosis of the *lungs* will also be treated of under the head of tubercular consumption; and the tuberculosis of the *nerve-centres* will form a part of the article on tubercular meningitis.

Blood-vessels are the seat of tuberculosis very frequently, inasmuch as their walls are the main receptacles for the deposit of the bacilli and tubercles in acute miliary tuberculosis. It originates along the finest ramifications. In very rare chronic cases, larger blood-vessels are affected, and may give rise, by weakening the elasticity of the walls, to aneurisms.

Symptomatology.—If we are again to characterize in a few words the nature of the tubercular infection, the process will be described thus: Through inhaling the dried and pulverized sputum of the consumptive, or through a local tubercular deposit undergoing disintegration and absorption, the bacilli are admitted into the circulation. That admission takes place through the lymph-ducts or the blood-vessels, mostly of the smallest size. But the largest vessels also have been known to be the direct carriers of the poison,—for instance, the thoracic duct, in a case of Ponfick, and large arteries and veins (Weigert) which become adherent to and perforated by neighboring caseous tubercles. If but little morbid material be admitted, or but little in repeated doses, the result is chronic tuberculosis or isolated tubercles in a gland, bone, joint, or nerve-centre; if there be much at a time, the result is acute miliary tuberculosis. A predisposition may be created under the influence of serious diseases, extensive suppurations, debilitating causes of every description, overcrowding and impaired health in cellars, factories, schools, nurseries, orphan asylums, prisons, and barracks, and by a number of infectious diseases which are eminently dangerous to the structure of the respiratory mucous membranes, such as measles and whooping-cough.

General tuberculosis has no such distinct symptomatology of its own as many of the other infectious or contagious diseases. Its localizations are so numerous that the individual cases exhibit a great variety of symptoms. Under the heads of the different organs, in the future essays and volumes of this work, the tuberculosis of the glands, the lungs, the meninges, the peritoneum, etc., will be discussed. Thus a few remarks must suffice here; they will refer mainly to the symptoms of the *chronic* and the *acute* form.

In both, the symptoms belonging to the general disease may be obscured by those of the organ solely or mainly affected. Still, there are a number of changes, mostly in the *chronic* condition, which, if they do not suffice to establish the diagnosis, render it highly probable. The majority refer to the state of the general nutrition.

In most cases this is defective. The children are thin and puny, or emaciate visibly, in spite of good and sufficient nourishment and fair digestion, and the absence of fever. Others, particularly infants fed on breast-milk, are, moreover, troubled with cough and elevated temperatures, but may lose no weight for many months; still, they arouse our suspicion by the above-mentioned symptoms and some unaccountable anæmia. The complexion in most cases is either pale or sallow; occasionally this result of anæmia and ill-nutrition alternates with a general or circumscribed flush on the cheeks, or is replaced by a cyanotic hue in those in whom the venous circulation is embarrassed by large glands or pulmonary disease. The sclerotic is bluish, the eyes moist or dry, and their expression languid or sad.

The skin is flaccid, wrinkled, and devoid of elasticity, dry, and liable to peel in very small scales. Perspiration and sudamina are found in such only as develop incidental attacks of fever or have a somewhat elevated temperature constantly. When anæmia has reached a rather high degree, there is œdema about the ankles or lumbar region (the locality depending on the position of the child, whether mostly erect or recumbent), and about the face when there is glandular swelling near the jugular veins.

The bronchi are mostly affected with catarrh, but frequently to a very slight degree only. Contrary to what might be expected in the presence of but few local pulmonary symptoms, there may be much dyspnœa, due to the multitude of miliary tubercles, or to the intensity of the hydræmic condition, or to the debility of the heart-muscle, or to all of these causes combined.

A frequent occurrence is the enlarged size of many of the accessible glands. Palpation reveals them round the neck, also in the inguinal regions, seldom in the axilla or abdomen. The tracheal and bronchial glands are often very numerous, and the dulness on percussion over their site is quite marked. It is particularly perceptible over the manubrium sterni, where, however, the persistence of the thymus gland may give rise to mistakes, and in the subclavicular regions. Here, too, the diagnosis may be difficult. For not only may the glands be swollen mainly on one side only, or more markedly than on the other, but the lungs, or one of them, may yield the same percussion-note in the presence of a chronic infiltration.

It is the *acute* form of tuberculosis which participates eminently in the characteristics of infectious diseases. It is always attended with fever and the appearance in many organs of numerous isolated miliary tubercles, which but rarely have the time to become confluent and form infiltrations. The latter, when found at autopsies, are mostly of older date than the miliary deposits. In these cases the infecting material spreads through the circulating lymph and blood from a single centre, which can be recognized in many instances. Caseous degeneration has long been suspected, and finally recognized, as the fountain-head of the generalized disease. The lymphatic glands, bronchial, tracheal, mesenteric, and retro-peritoneal, in their intimate relations with the lymph-ducts and the circulation of the blood, furnish the morbid material an easy road to the rest of the body. If that material consist of disintegrated cells and nuclei only, the result will be some process or processes of embolism, with local anæmia, inflammation, disintegration of tissue, or pyæmia; if it contain specific bacilli, miliary tuberculosis will follow. The most rapid course of the malady must be expected when the growing gland proliferates into the lumen of a vein. In this way, besides the glands, caries of the bones, tubercular arthritis, and purulent pleuritis or ulceration of mucous membranes will lead to the same end. Defective conditions of the latter, such as are the results of whooping-cough, measles, or typhoid fever, furnish, besides, ample opportunities for the admission of the bacillus from outside. After this has been accomplished, the formation of a tubercle is explained by M. V. Cornil[1] in this way: that bacilli penetrating into the tissue-cells give rise to a nutritive and formative irritation, exhibiting as its first result a subdivision of the cells. This process takes place in the cells of the connective tissue, the endothelia of the blood-vessels, and the epithelia. Besides, the presence of bacilli produces embolic processes in the capillaries, and gives rise to alterations in the walls of the blood-vessels and emigration of leucocytes. These again emigrate, and penetrate into the tubercles while in the process of formation.

The tubercles are either gray—in the very recent state—or yellow. Both varieties are mostly found together. They are met with in and on the liver, lungs, kidneys, intestines, pia mater, peritoneum, pleura, bones, dura mater, brain, pericardium, stomach, thyroid, but rarely about the genital organs and the muscles.

The order in which they have been here enumerated indicates their susceptibility and numerical importance. The thymus gland I have proved to be also affected more frequently than was known before. Indeed, I have found an instance in which that body was the primary seat of the disease. It is probable that it will be found to be a more frequent abode of tubercular deposits than the choroid, retina, and iris.

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[1] Études exp. et clin. sur la Tuberculose, publ. sous la dir. de M. le Prof. Verneuil, Paris, 1887, fasc. i.

The very multitude and variety of organs in which the tubercular deposits gain a footing and undergo further development, explain the difference in, and the multiplicity of, the symptoms. The fever and some tumefaction of the spleen are common to all acute infectious diseases. Indeed, the latter is enlarged though there be no local tuberculosis of the organ either on the surface or in its tissue, and may, under these circumstances, be mistaken for that of typhoid fever.

When the respiratory organs are the principal seat of the tubercular infection, the symptoms do not always correspond with the extent of the lesions. As, however, this subject will be treated of more extensively in a subsequent paper, an outline only of the changes and symptoms connected with the pulmonary localization of general tuberculosis will be given on this occasion. There is bronchitis, sometimes quite extensive, with all the physical signs of hyperæmia and thickening of the mucous membrane, and expectoration which, when brought up at all, contains fewer bacilli than are found in tubercular consumption proper. Blood appears but rarely, except in the latter form. Cough is not so frequent as the pulmonary and bronchial changes would lead us to expect, because of the frequent prevalence of the brain-symptoms. There is sometimes a high degree of dyspnœa, particularly in those cases which exhibit cardiac debility at a very early period of the malady. Respiration is often quite rapid (without much apparent dyspnœa), though there may be but little solid infiltration. Indeed, percussion yields often but a negative result even in advanced cases. Not infrequently the soft friction-sound of accompanying tubercular pleurisy is more evident than are physical symptoms belonging to the lungs, with the exception of those instances in which an acute and extensive pneumonia takes the place of the multiple, but small, alterations.

Encephalic tuberculosis and tubercular meningitis will form the subject of a special article. Here it may be mentioned only that the principal symptom of an infectious disease—viz., fever—is often absent in these forms. Indeed, though the disease is of the most serious nature,—the localization in the brain giving rise to retarded and irregular pulse, vomiting, peripheral contraction, and paralysis of a multitude of muscles in different organs, to the suppression of secretions, and even to convulsions and coma, —the temperature of the body is not liable to be raised before the very end of life.

Diagnosis.—The diagnosis of miliary tuberculosis is by no means easy. Both in the adult and in the child it has often been mistaken for typhoid fever, and *vice versa*. It is true that in miliary tuberculosis there is "often" pallor and cyanosis, slow and intermittent pulse, and dyspnœa without objective symptoms; but these are the cases which offer no difficulty, as a rule. The most serious cases are exactly those in which the diagnosis is apt to go astray. Typhoid fever in the young is by no means the regular strait-jacketed disease, as some text-books still insist upon describing the same disease when in the adult; its temperature does not follow the exact curve claimed in print, the daily curves are sometimes double, the temperatures are either high or low through the whole course of a case, there are, or may be, bronchitis, splenic tumor, diarrhœa, roseola, or chills. Now, all these symptoms are found in acute miliary tuberculosis as well. Even Ehrlich's diazo-test of the urine is not conclusive; indeed, it has long been acknowledged that, in the differential diagnosis between the two, it is unreliable. Now, it is true that in miliary tuberculosis the bacillus may be found in the blood, in the expectoration if there be any, or in the stools, or miliary deposits may be discovered in the choroid or retina. But there will be many cases in which even the most expert diagnostician will fail. Indeed, even as well-marked an affection as tubercular meningitis may be difficult of diagnosis from typhoid fever, particularly on account of the fact that genuine meningitis (not to speak of meningeal symptoms) may be an actual complication of typhoid fever.

The diagnosis of tuberculosis from a malarial process is not always made quite readily. The latter may linger long; there may be no fever observed or existing; or an occasional rise of temperature, lasting from a day to a week or more, is noted, and occasional apyrexia extending over days or weeks. There is now and then thirst, dry and hot skin, perhaps no chill, but increasing emaciation, anæmia, and listlessness. The same symptoms will be found in chronic tuberculosis, in which the local symptoms may be very indefinite or obscure. Even feverish cases of tuberculosis may not be quite conclusive, in the absence of positive local symptoms. In tuberculosis exacerbations of temperature take place mostly towards the evening, those of malaria frequently in the forenoon. But "frequently" and "mostly" yield no diagnosis in an obscure individual case; it must not be decided by a presumable average any more than by the result of questionable treatment. For the assertion that quinine will relieve the fever of malaria, while it is ineffective in that of tuberculosis, must be received with many grains of salt.

Prognosis.—The prognosis of tuberculosis is always grave. The termination of the acute miliary form is almost always fatal. The large number of recoveries sometimes reported does not agree with the experience of those who see their cases from beginning to end. A single visit does not always suffice to make the diagnosis; on the contrary, localized miliary tuberculosis may often be presumed to exist without a sufficient cause. Thus only can I explain the fact that one of the foremost and most conscientious consulting physicians in the American profession gave it as his honest conviction that one-sixth part of all cases of tubercular meningitis got well.

The chronic form may recover. Even in autopsies made on persons who died of miliary tuberculosis we are apt to find localized tubercles so hardened and encysted that they at least cannot be accused of having given rise to the acute infection. Besides, the finding of solitary tubercles in the lungs (or occasionally other organs) in the post-mortem examinations of people dying of miscellaneous diseases is more than an occasional occurrence. It is quite frequent in the adult, and not unusual in the bodies of children of ten or twelve years. Thus, indeed, chronic tuberculosis may heal, temporarily or permanently; but still the prognosis in every case which has been diagnosticated ought to be rather worse than merely guarded. That rule is more imperative in the young than in the adult; for it is in the former that, in consequence of the greater activity of lymph and blood circulation and absorption, a universal infection originating from a local cause is more easily accomplished. Many organs are affected at the same time. In one hundred and sixty-two cases of tuberculosis, Lorey found twenty-two of acute miliary infection, sixty-two of tuberculosis in the bronchial glands, eighty-three in the lungs, twenty-nine in the brain, twenty-one in the bones, and twenty in the spleen.

Treatment.—Tuberculosis cannot be *prevented*, or limited, under our political and social circumstances, by the prohibition of marriages of tuberculous people, or the separation of children from their parents, or the removal of phthisical workmen from their shops or factories. Nor would such measures be successful to such an extent as has been presumed by hasty reformers. For, indeed, the danger of the propagation of tuberculosis from person to person by respiration is but slight; no current of air is capable of removing bacilli or spores from a moist surface such as the mucous membrane of the bronchial tubes or the surface of a cavity. For the same reason, neither the fæces expelled from a tuberculous intestine nor the urine eliminated from diseased urinary organs can often transmit the malady.

The bacilli conveying the disease are far from being ubiquitous. They have a higher specific gravity than air, water, or even pus; their growth is slow, and easily interrupted by the presence of putrefaction and other schizomycetes endowed with rapid proliferation; they require a temperature of at least 30° C. (86° F.), which they cannot find permanently except in the animal body; and it is in the latter only that they find their nourishment. Here they develop and multiply, and become dangerous when, after leaving it, they are preserved in a dry state. Even thus, a certain length of time—perhaps six months—destroys their efficacy; and, though one-seventh part of mankind die of tuberculosis, mostly of the lungs, it is evident that the expectoration of months and years becomes dangerous in relatively but rare instances. In order to be so, the sputum must be dry, finely distributed, and inhaled; for, though tuberculosis may be found in most tissues and organs, the lungs are the principal inlet and outlet. Even here, however, the invasion into the system is not easy. For its principal locality must be the very finest ends of the bronchial ramifications and the air-cells; if deposited in the larger bronchi, the bacilli would be readily expelled by the secretion of the muciparous glands and the uninterrupted activity of the ciliated epithelium. Still, it is the sputum, dry, finely pulverized, and entering the lungs or coming in contact with sore surfaces, which yields the principal danger, and the main preventive measure is its disinfection or destruction before it can do any harm.

Though the bacillus is long-lived and not easily destroyed, there are a great many ways of preventing the disease from spreading. The best preventive is a healthy mucous membrane. A simple catarrh may afford an inlet, and ought, therefore, not to be made light of in a family or surroundings in which tuberculosis has found a home. The bronchitis of measles and whooping-cough, rendering the surface amenable to infection, requires care; nothing can be more dangerous, therefore, than the supercilious indifference too often exhibited by practitioners dealing with these diseases, as unworthy of their attention, because they are self-limited in their course of weeks or months. As the communicability of the bacillus is very great when it is in a sufficiently dry state to be inhaled, the expectorated substances must not be permitted to be preserved on towels or handkerchiefs, or to remain on bedding and floor, or spoons, or vessels, or whiskers from which the innocent kiss of the child will be poisoned. The sputum must be deposited in a moist vessel, and soon removed; in the sink and sewer, or on the field with the rest of the sewerage, which will render the bacillus of tuberculosis innocuous by moisture or destroy it by putrefaction, it will do no harm. The patient will protect himself from auto-infection by remembering that his own sputum, when dry, is a weapon turned against himself. Besides, a thorough disinfection must be applied to clothing and furniture by excessive heat, great care exercised in the selection of the school, companions, and nurses, and the room thoroughly disinfected in which a consumptive patient has lived or died. Von Esmarch recommends to rub down the walls, and the wood of the furniture, with bread.

Much may be done by the enforcement of public hygiene. Among the working-men or -women of a factory ten per cent., more or less, are consumptive. Their sputum is expectorated on floors and furniture, will get dry and pulverized, and inhaled. Thus the germ is carried over the community, old and young. From the tailoring establishments large and small, ready-made clothing-shops, etc., the material to be worked up is given to the tens of thousands of men and women in whose dingy tenements tuberculosis, diphtheria, and other contagious diseases are indigenous. From these they infect the community. This frightful fact is sufficient to discourage the most hopeful philanthropist; it proves again the embarrassments and dangers of our social conditions, and the great difficulties an enlightened public hygiene will have to overcome.

That no child ought to drink milk without its being thoroughly boiled, goes without saying, when it is understood that tuberculosis is a frequent disease of the cow, and both its milk and its meat may become the cause of infection; the former, however, only (though there are those who do not agree with this statement) when the udder participates in the disease, which is of common occurrence, though difficult to diagnosticate; the latter but rarely, because the muscular tissue is almost exempt from tuberculosis. Thus, indeed, the danger is reduced to a minimum when the meat is thoroughly heated, and the organs of the animal most subject to the invasion of the disease (such as liver, thymus, lungs, and viscera in general) are excluded from the bill of fare.

The preventive extirpation of tubercular glands has been recommended and practised extensively. It is mainly the glands of the neck which are accessible. They are infected by every irritation of the head, face, mouth, and nares. In all of these parts primary tuberculosis is not frequent at all, but the invasion of bacilli and their transmission from the superficial sores to the glands is at least a possibility. At all events, however, the larger number of the tumefactions owe their origin, not to the specific bacilli, but to an irritation of a less dangerous kind. Now, when caseous degeneration takes place in a gland swelled by any cause whatever, though not of a specific order, the absorption of the detritus may lead to embolic processes; if, however, the caseous gland contains the bacillus, tuberculosis will follow absorption. In every case, then, the extirpation is advisable. But the final result of every such operation is jeopardized by the fact that, generally, we have not to deal with a single isolated gland, but with a great many. For this reason the operation is liable to fall short of its aim, because of the impossibility of removing everything morbid. It is particularly in young children that this ill success has been experienced.

Cold abscesses, of tubercular nature, must be treated according to their seat and origin. Those of the subcutaneous tissue may be incised, their walls scooped out, disinfected, and either drained or filled with iodoform gauze. Now and then the advice has been given to wait for a spontaneous rupture of the surface, but incision and antiseptic treatment are preferable. Those connected with bones, and sometimes so by long and sinuous fistulae, require operations of greater magnitude, extending to and including the bones.

The treatment of tubercular disease of the bone must be local, though in many cases it be as unpromising as general medication. In tubercular spondylitis neither the operative nor the expectant nor the medicinal plan is very successful. Coxitis is more amenable to the former, and its results are more favorable. The same can be said of the tubercular affection of the knee-joint, the ankle-joint, and the bones of the tarsus. The methods of the operation cannot be identical; whether resection, the scoop, or ignipuncture is selected must depend on the extent and location of the lesion. After the operation, and sometimes without it, iodoform treatment has been found beneficial. At all events, the diseased capsular ligaments must be effectually removed.

Whatever aids in fortifying the tissues against the invasion of bacilli must be looked upon as welcome, inasmuch as the treatment of the established disease is among the most unpromising. For the effect of antifermentative or antibacteric remedies when introduced into the animal organism unfortunately does not correspond with that produced in the test-tubes. G. Cornet publishes a series of experiments[1] made on one hundred guinea-pigs and ten rabbits previously infected with tubercle-bacilli, either subcutaneously, or through inhalation of the finely-distributed material. The remedies employed were tannin, acetate of lead, garlic, pinguin, sulphide of hydrogen, menthol, corrosive sublimate, creolin, and creasote. The latter diminished the secretions, but none of them, though introduced in large doses and for long periods, exhibited any antibacillary effect. Nor did altitude have any effect, for some of the animals infected in Berlin were sent to Davos in Switzerland, unsuccessfully.

The antibacteric medicines which thus far have been of most service to operative surgery cannot be expected, with our present knowledge, to be made useful in the treatment of chronic or acute general tuberculosis. The subject, however, will be discussed more extensively in the article on pulmonary tuberculosis. The very necessity of emphasizing the strengthening of the system against the inroads of the disease, indicates the comparative powerlessness of the body against its devastations when once begun.

My experience with arsenic in pulmonary phthisis, as a tissue-builder and nutrient, leads me to recommend it in the other forms of localized and universal tuberculosis. Of phosphorus I have not seen so much in this direction, but its effect is the same, and its superior efficacy in the chronic and subacute diseases of the bones ought to justify its administration in behalf of the system threatened with tuberculosis. As the feeble connective tissue requires arsenic and phosphorus, so the incompetent heart-muscle needs its own tonics; for digitalis, spartein, and caffein, while stimulating the heart into supplying the provinces of the body with more blood, render the same service to the heart, and thus improve the general nutrition. When acute tuberculosis has made its appearance, the medicinal treatment can be symptomatic only. The general principles of therapeutics must be applied here as elsewhere: antipyretics, narcotics, and stimulants will find their places according to the most prominent symptoms. Unfortunately, the disease, when fully established, leaves the practitioner no better opportunities than to fulfil the indications suggested in the interest of euthanasia.

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[1] Zeitsch. f. Hygiene, 1888, v., 98–133.

SYPHILIS.

By ABNER POST, M.D.

Syphilis is known to us as a chronic infectious disease, due in all probability to a specific micro-organism. In order that a previously healthy individual may acquire the disease it is simply necessary that the blood, or such discharges or secretions from a syphilitic individual as contain the syphilitic contagium, shall be brought into contact with an abraded surface or at least a surface capable of absorption. Such discharges applied to an abraded surface on a child will produce the same effect as in the adult: the child will acquire syphilis; and its first manifestation will be a primary sore or chancre at the spot of inoculation. Syphilis thus inoculated upon a child will run a course practically identical with the disease in the adult, modified only by the peculiarities of the infant organization. Infants may also be syphilitic by direct inheritance from syphilitic parents, in which case the general manifestations are never preceded by a chancre.

Inherited syphilis manifests itself in infancy with variable severity, conforming more or less completely to the state of the disease in the parents. It is fatal in a large percentage of the cases. Of the children who survive, many reach a condition of apparent health, though many bear through life the marks of their inheritance. The disease runs a course which resembles closely the secondary manifestations in the acquired disease in adults. Probably there is no single lesion that occurs in adult acquired syphilis that may not occur in hereditary syphilis, which possesses in addition features peculiarly its own. Its contagious properties fully equal those of the acquired disease. It manifests its existence by various lesions of the skin, and the survivors usually gain a condition of apparent health after the lapse of two years, more or less. The victim of hereditary disease is also subject to subsequent outbreaks which resemble the later so-called tertiary manifestations of acquired syphilis.

Infantile syphilis must, then, be considered under the two forms of (1) hereditary and (2) acquired disease, and for convenience of description the late hereditary forms of the disease will be separately considered.

HEREDITARY SYPHILIS.

Syphilis derived from one or both parents and existing already in the infant at its birth is known as hereditary, inherited, transmitted, or congenital syphilis. The term infantile syphilis has a wider significance, and ought not to be used when it is intended to designate simply the inherited disease. Bœck, of Christiania, restricts the term congenital to those cases in which the disease is supposed to come from a mother contaminated during pregnancy.

The question of the origin of hereditary syphilis, whether from one or both parents, has such direct bearing upon prophylaxis as regards future children, and upon certain questions regarding the rearing of existing children, that any one who is interested in the children themselves must of necessity be interested in the etiology of the disease.

Unfortunately, there is not perfect agreement on the questions concerned as discussed by different writers, nor is there perfect accord in the recorded facts. The matter appears exceedingly complicated. Undoubtedly the apparent difficulty exists because our knowledge of the laws of heredity is imperfect. An extended discussion would be out of place here. It is only necessary to express as briefly as possible the opposing beliefs and the reasons for holding them. It will then be possible to formulate a working schedule of known and probable facts which shall serve as a guide for our actions.

Taking up at first the connection of the father, we are confronted at the very outset with two entirely different sets of facts,—one of which goes to show that syphilitic fathers may beget healthy children, the other that children are brought syphilitic into the world when the father is known to be syphilitic and the mother presents no sign of the disease, though carefully watched through a long series of years.

As regards the first series of facts, it is a matter of frequent occurrence for syphilitic men to beget healthy children, some of these men afterwards showing evidence of the persistence of the disease in their own bodies by the recurrence of symptoms. Cases are even recorded in which men bearing evidence of recent syphilis at the very moment of conception are said to have begotten healthy children. Fournier reports a series of eighty-seven cases under his own observation, in which syphilitic men have married, have communicated nothing to their wives, and have a total of one hundred and fifty-six children, all of them healthy. But although a syphilitic father does not always of necessity transmit the disease to his offspring, and although the possibility of such transmission is denied by certain eminent syphilographers,[1] a very large number of cases are on record in which the observer believed that the disease had been so transmitted.[2] Fournier quotes a case from Parrot, observed by him under special conditions "which leave no possibility of error." "A young man married with syphilis in full activity. He had two children, who both presented undoubted symptoms of hereditary syphilis. Now, their mother, closely watched over, minutely examined from time to time since her marriage, has never presented and still does not present any suspicious symptom. Without doubt she remains entirely exempt. Kassowitz does not hesitate to say that the paternal inheritance of syphilis may be ranked among the best-established scientific facts, and that the continued opposition of unbelievers can no more change it than, for example, can the protective power of vaccinia against small-pox be rendered doubtful because annually whole libraries are written and printed against it."[1]

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[1] The possibility of transmission from the father alone is positively denied by Cullerier, Oewre, and Sturgis.

[2] Among the authors who have reported such cases are Ricord, Trousseau, Diday, Cazenave, Hutchinson, Bärensprung, Bassereau, Parrot, Lancereaux, Kassowitz, and Fournier.

The power of transmission is greatly weakened by the use of mercury, and is sometimes absent while the disease is in a quiescent state, and is usually if not always finally extinguished by the effects of treatment or the lapse of time, or by both, and one of these causes will be found in operation in most of the cases in which the child of a syphilitic father is healthy. The chief danger for the offspring from a syphilitic father lies in the probability that he will infect the mother. The offspring will then have a double syphilitic parentage, a condition more disastrous than syphilis in either father or mother alone.

Maternal.—In the majority of cases of syphilis in infants the mother is manifestly syphilitic. Usually she has taken the disease from the father,—occasionally she has given it to him,—so that both parents are syphilitic, and it is impossible to determine the part played by each in the transmission of the disease to the infant, but there are certainly examples that show that the mother alone, the father being unaffected, if her disease is active at the time of conception, may transmit the disease to her offspring, and those cases in which the mother alone is responsible for the disease in her offspring are certainly much more numerous than those in which the father alone is responsible.

An important question arises in regard to the women who give birth to syphilitic children while remaining themselves apparently healthy: Are these women really healthy, or are they cases of latent syphilis? The observations that go to establish the health of these women are very numerous. In 1874, Kassowitz published a monograph in which he strongly upheld the health of this class of women. He gave careful statistics of the Vienna Foundling Asylum, where out of four hundred children with hereditary syphilis one hundred and sixty had healthy mothers, one hundred and twenty-two had syphilitic mothers, and in the remainder of the cases the condition of the mothers was not known. In addition he gave seventy-six cases of his own, in forty-three of which the mothers were healthy, in twenty-three both parents were syphilitic, and in ten the mothers only were diseased. In 1884,[2] Kassowitz reviewed the literature on the subject for the ten years succeeding his first monograph. In this second communication he stated that a large number of the mothers whom he had previously reported healthy had remained under his observation, and that in none of them had a single suspicious symptom shown itself. The communications of others on the subject he divided into three groups: (1) those of the general practitioners and children's doctors, (2) those of the obstetricians, (3) those of the syphilographers. The observations of the general practitioners he considers as of special value, as they are able to follow their patients very closely and for a long time. As an example, he gives a case of his own. The husband was infected four years before marriage. In spite of a moderately energetic treatment, a syphilitic child was born during the first year of marriage, and speedily died. Further treatment of the father was followed by the birth of a child which had reached the age of nine years in good health. Six years later another child was born, which at time of narration had reached the age of three years in health. The wife discovered the cause of her first baby's death and watched herself carefully, but neither she, Kassowitz, nor the obstetrician called in for the subsequent deliveries could discover any sign of disease. The husband alone was treated. Such cases are seldom published by physicians, as they look upon the matter as settled. As a further example of such observation, Rosenburg[1] publishes a case in which the mother of syphilitic children was watched over for a series of years by the same physician who at last cared for her during a fatal hereditary phthisis, but she was always free from every syphilitic appearance.

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[1] Jahrbuch für Kinderheilkunde, 1884. p. 69.

[2] Ibid., p, 52.

The evidence of the obstetricians is of an entirely different character, as it embraces for the most part observations in lying-in hospitals, where patients are under observation but a relatively short time. But the shortness of the time is compensated for by the large number of observations and by the opportunity of making the most careful examinations, and by the fact that during the last months of pregnancy syphilitic manifestations in the region of the genitals often reach colossal dimensions. Three reports from large obstetrical clinics have appeared,—from Berlin, from Dresden, and from Munich. Mewis[2] reports, from the clinic of Winckel, of Dresden, one hundred and nine syphilitic children from one hundred and eight mothers who presented no sign of syphilis. Anton[3] reports, from the clinic of Gusserow, that in thirteen months seventy mothers gave birth to syphilitic children. Of these women fifteen were certainly free from syphilis. V. Hecker[4] in fifty-three new-born syphilitic children could find no trace of syphilis in the mothers.

A long series of observations is also reported from men more particularly devoted to the study of syphilis, in which the mothers of syphilitic children born of syphilitic fathers have presented absolutely no sign of syphilis, though examined with the greatest care. In addition to such observations by previous authors may be cited the more recent cases of Taylor[1] and J. Nevins Hyde.[2] Fournier[3] reports fourteen cases of that character, in which he affirms the mother's freedom from disease. Neumann also reports a case in which it was impossible to discover the slightest sign of syphilis, though the woman was kept under observation for six months.

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[1] Deutsche Med. Wochenschrift, No. 31, 1883.

[2] Mewis, Ueber Schwangerschaft, Geburt und Wochenbett Syphilitischer, etc., Zeitschrift für Geburtshilfe und Gynaecologie, Band iv. S. 10, 1879.

[3] Anton, Ueber hereditäre Syphilis, Inaug. Diss., Berlin, 1880.

[4] Hecker, Beobachtungen und Untersuchungen an der Gebäranstalt zu München, 1859–1879, München.

Observation shows that these women possess one marked peculiarity in an apparent immunity as regards liability to contract syphilis. This fact was brought into special prominence by Mr. Colles, of Dublin, who stated that he had never seen or heard of a single instance in which a syphilitic breast-fed child, deriving the infection of syphilis from its parents, had caused an ulceration of the mother's breasts, whereas very few instances have occurred where a syphilitic infant has not infected a strange hired wet-nurse who had been previously in good health.[4]

This observation, enunciated as a principle, is known as Colles's law. The observations that tend to invalidate the law of Colles are few, and the immunity of the mother is usually admitted. Three possible explanations of this immunity have been advanced: that the mother, notwithstanding her apparent health, is actually syphilitic, having been infected (1) by the father, or (2) by the child through the utero-placental circulation, or (3) that, though not actually syphilitic, she has undergone some imperfectly-understood transformation which renders her for the future insusceptible to actual inoculation.

This question of the syphilis of the mother is but the question as to the possibility of inheritance from the father alone. An example has already been given of the observations that seem to establish the mother's health. On the other hand, Keyes reports a case in which he found, at one examination of an apparently healthy woman just after the birth of her syphilitic child, a few doubtful macules on the skin, and some "small but beautifully characteristic mucous patches upon the throat and inside the mouth." The husband was known to be syphilitic. The mother's symptoms passed away, and nothing further was found upon her. She subsequently bore two syphilitic children. Had he not seen the mother at that particular time, Dr. Keyes would have felt certain that she had no syphilis, and that she was another example of a woman giving birth to syphilitic children and remaining healthy herself. His observation shows that some, at least, of these apparently healthy women are really syphilitic.

The doctrine that syphilis may be transferred to the mother from a syphilitic fœtus finds very wide but by no means universal acceptance. The process is known as *choc en retour*, retro-infection, or syphilis by conception, and is invoked to explain those cases in which the wife has become syphilitic without a discoverable chancre. Fournier believes that there is a large class of cases in which the woman presents no initial lesion, in which the husband has no contagious lesion, and in which the disease first shows itself in the wife during pregnancy. So long as the woman remains unimpregnated she is free from syphilis, but when she becomes pregnant syphilis breaks out. Fournier can find no other explanation for these cases than infection of the mother by the child in utero. Other authors find it difficult to believe that syphilis can be conveyed to the mother from the fœtus through the placental circulation as syphilis, but still believe that the syphilis of the child is not without effect, and that the mother receives the disease in a modified form which renders her for ever after insusceptible to syphilis.

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[1] Archives of Clinical Surgery, September, 1876.

[2] Archives of Dermatology, April, 1878.

[3] Syphilis and Marriage, 1880.

[4] Practical Observations on the Venereal Disease, 1837, p. 285.

Post-Conceptional Syphilis.—Whether a pregnant woman who becomes syphilitic during her pregnancy conveys the disease to her child in utero, is a closely-related question. That such conveyance takes place is strongly affirmed by some of the most reliable authorities. It is certain that the woman may abort or miscarry, but that the untimely-delivered product of conception is actually syphilitic is not yet conclusively shown. It is unsatisfactory to appeal to analogy as to the power of the placenta to act as a filter in other diseases, as the results of observations are not uniform. Neumann[1] reported in 1885 observations on twenty cases of post-conceptional syphilis: of the twenty children resulting, five were syphilitic and fifteen non-syphilitic. One of these children was infected by its mother at the age of seven months. The conclusion best adapted to reconcile existing observations is that in the majority of cases of post-conceptional syphilis of the mother the fœtus then in utero is protected, but that post-conceptional syphilis is transmitted to the fœtus in a few cases. Whether the mother is affected in the beginning or in well-advanced pregnancy makes no difference in the liability to transfer the disease to the fœtus.

If such an explanation is correct, it would seem, and certain observations hereafter to be mentioned bear it out, that, while the placenta is normally a filter which prevents the passage of the syphilitic germ either from mother to child or from child to mother, under the influence of disease it occasionally loses its control and allows intra-uterine infection.

Conclusions.—The following summary may be accepted as embodying the principles which should control us in our practical relations with our patients.

It is to be expected that children will be syphilitic by heredity when one or both parents are recently syphilitic at the moment of conception.

When both parents are syphilitic at the period of conception, there is greater probability that the children will be syphilitic than if one parent alone has syphilis. At the same time, the transmission of syphilis to the offspring is not inevitable when the parents, one or both, have the disease. The more recent the disease, the greater the probability of its transmission, and the more probable that the disease in the offspring will assume a severe form.

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[1] Med. Jahrbüch. d. Ges. d. Aerzte, Wien, 1885, Archiv f. Kinderheilkunde, vii. 222.

The aptitude to transmit the disease decreases spontaneously, in many cases, with the lapse of time. The influence of mercurial treatment of the parents upon the health of the offspring is much more certain than that of time alone. A pregnant woman who is syphilitic, whether her syphilis was acquired before or after conception, should be thoroughly treated, to avoid disaster to the child.

The earliest manifestation of syphilis upon the product of conception is shown by abortion. The disease is so common a cause of miscarriage that, when premature labor has repeatedly occurred, suspicion should always be excited as to the previous condition of the parents, so that succeeding children may be rescued by the proper treatment of one or both parents. Mercurial treatment has been accused of causing abortions. Excessive use of mercury may possibly cause abortions, but it is certain that well-directed treatment by mercury has only beneficial results in retarding abortions and causing living children to be brought into the world. Abortions are much more frequent when the woman is in the early stages of syphilis than later. It has been considered that in many of the incompleted pregnancies the abortion is due to the anaemia and debility of the mother rather than directly to syphilis. Some of the more recent studies of the placenta would indicate that abortion in the early cases is the result of disease of the maternal placenta.

The aborted fœtus of a syphilitic woman is usually macerated, but undeniable lesions of syphilis must be found in the child itself before it can be affirmed that a macerated fœtus is syphilitic. Women afflicted with other diseases than syphilis abort and bring forth macerated children, though the large majority of macerated fœtuses are syphilitic. In an examination of still-born children and children who died soon after birth, Birch-Hirschfeld found that of the macerated fœtuses seventy per cent. showed unmistakable signs of syphilis.[1] In such children the skin usually shows no sign of syphilis; it is of a livid purple color and is easily detached. Large bullæ may be found on the palms and soles. It is only by careful examination of the bones that syphilis can be certainly determined to be present.

Syphilis of the placenta can hardly be said to be known in all its details. A very complete work on the subject, based on the study of about three hundred placentas, was published in 1885 by Zilles, from the obstetrical clinic of Prof. Säxinger, of Tübingen. His conclusions, which have not been long enough before the public for thorough examination, are practically as follows:

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[1] Archiv der Heilkunde, No. 16, 1875, S. 172.

1. There is a placental syphilis which can be diagnosed microscopically in many cases.

2. Placental syphilis occurs usually in connection with fœtal syphilis, but not, as Fraenkel states, only with fœtal syphilis. Placental syphilis can coexist with a syphilitic mother and a healthy child. This occurs in those cases in which the mother is infected during the course of the pregnancy and gives birth to a healthy child.

3. The placenta may be diseased in its whole thickness, or in the maternal or in the fœtal part alone.

a. If the mother is infected at the same time with the impregnation, we find fœtal syphilis, and the placenta usually more or less diseased in all its parts. In such cases the vessels of the cord are generally diseased.

b. If only the sperm-cell is the bearer of the syphilitic virus and the mother is not infected, we find fœtal syphilis, and for the most part only in the placenta fœtalis and the cord; yet can the process, as Zilles has seen in isolated cases, encroach upon the placenta materna and so lead subsequently to an intra-uterine infection of the mother.

c. (a) If the mother was infected shortly before the conception, and if, the disease not being yet constitutional, the woman is impregnated by a healthy man and during the pregnancy is subjected to antisyphilitic treatment, then a healthy child will be born. The placenta shows itself in this case diseased only in the maternal part. (b) If the woman was infected a long time before the impregnation, it often occurs that the placenta materna alone is diseased; yet, by the progress of the disease from the placenta materna to the placenta fœtalis, the whole placenta may become diseased, and, as a consequence, the fœtus also become infected, if it has not already died in consequence of the disturbed circulation in the placenta.

d. If the mother, pregnant by a healthy man, is infected during the course of the pregnancy, then we find immunity of the fœtus generally, but the placenta materna is always, though often slightly, diseased.

Zilles does not believe, contrary to the teaching of Fraenkel, that a placenta from a syphilitic woman can be free from all syphilitic manifestations, unless the mother was infected so short a time before the birth that an outbreak of constitutional symptoms had not yet manifested itself up to the time of the birth.

If these views of Zilles stand the test of further investigation, they will accomplish much towards simplifying the disputed points in the matter of heredity.

Hydramnios is an occasional effect of syphilis; that is, hydramnios may result from several causes, of which syphilis is one. This accident of pregnancy is constituted by the excessive accumulation of fluid in the cavity of the amnion. The liquor amnii comes from many sources, one of which is the fœtal organism. The liquids in the umbilical vein when submitted to strong pressure traverse the vascular walls and appear in the amniotic cavity. Hydramnios is for the fœtus what ascites is for the adult when brought about by disturbance of the portal circulation. The liver is one of the organs most frequently attacked in the fœtus by syphilis. Circumscribed or diffused cirrhosis of the liver is already well advanced in certain subjects during intra-uterine life. The obliteration of veins which is its consequence increases the pressure in the umbilical vein, and hydramnios ensues. This result is, however, rare in comparison with the number of diseased livers; it is necessary that the lesion should be well advanced, and perhaps other conditions are also necessary.

Pathology.—In children who die at an advanced stage of intra-uterine life are found certain pathological tissue-changes in the viscera and in the bones. The same changes, but less marked, occur in children who die of inherited syphilis after birth. These visceral changes are practically the same as those which occur in acquired syphilis, but are much more common in the inherited disease. Diffuse interstitial hyperplasia is much more common in inherited disease than circumscribed gummy tumors. In the youngest fœtus the changes in the bones are most marked, and so common that the bony changes may almost be deemed necessary to establish syphilis in the fœtus.

Before proceeding to give the clinical features of congenital syphilis, the pathological changes which are found in the viscera of heredito-syphilitic infants will be briefly enumerated. These changes are more or less constant. It may be assumed that the infant is always affected in some internal organ: he certainly suffers much more frequently than does the victim of acquired syphilis.

Liver.—Gubler gave in 1852 a description of the liver as altered in syphilitic infants, which has served as a basis for all subsequent descriptions. The affected liver is always larger than in the normal condition. Its surface often presents thickenings of the capsule of Glisson. The hepatic tissue is harder and more elastic than normal: it rebounds when a piece is dropped upon the table. It presents the yellow color and the semi-transparency of flint. Gubler also described small white granulations, which he compared to grains of wheat, and which are scattered throughout the parenchyma. According to Cornil, these small granulations are formed by an accumulation of embryonic cells in the spaces which separate the hepatic acini. The hepatic acini, in the normal state, are in contact except at the prismatic spaces which are formed by their union, in which spaces the capsule of Glisson forms an envelope to the afferent portal vessels of the lobule. It is in these spaces that the round lymph-cells form and collect into small lobules representing microscopic gummata. The portal veins also present thickened walls with newly-formed cells in their external tunics.

Local peritonitis often accompanies this disease of the liver.

The gummata of the liver, resembling those of the adult, are also found in very young children affected with hereditary syphilis.

Spleen.—According to Parrot, the spleen is after the bony system the part most often attacked. Cornil[1] states that it is always hypertrophied in hereditary syphilis. The capsule is thickened and inflamed, and the splenic tissue is harder than normal. According to Dr. Gee,[2] the spleen is enlarged so that it can be felt during life in about half the cases of congenital syphilis. In about a quarter the enlargement is really great. Sometimes, in addition to the enlargement of the spleen, there is enlargement of the liver and of the lymphatics. The majority of cases of great enlargement die. The degree of splenic enlargement may be taken as an index of the severity of the cachexia, with the exception that once enlarged the spleen may remain enlarged for years. Dr. Gee found the enlargement to be a simple hypertrophy with considerable thickening of the capsule. In Dr. Barlow's[3] case there was simply slight enlargement with hardness: there was no reaction with iodine, and there were no gummata.

Pancreas.—In 1875, Birch-Hirschfeld directed special attention to changes in the pancreas. After his attention was directed to that organ, he examined the bodies of twenty-three new-born children that bore unmistakable evidence in the bones of syphilis, and in thirteen cases found the pancreas more or less altered. Of these twenty-three cases ten were macerated, and among these but two showed disease of the pancreas, while in the thirteen remaining cases, which died either during birth or soon after, the pancreas was diseased eleven times.

In the most marked cases the organ was much enlarged, its weight doubled, its tissue firm. On section it presented a glistening white appearance, and resembled more a fibroid than glandular structure. Microscopically the interstitial connective tissue was found greatly increased, especially between the larger lobules. This extreme alteration was found in seven cases. In six the changes were less marked. In some cases a part of the organ, especially the head, was decidedly changed while the rest was less markedly altered. Hirschfeld remarks that this interstitial change in the pancreas bears the fullest analogy with the interstitial changes in other organs, especially the liver, which have long been recognized as the product of syphilitic infection, and, while it is not constant, it comes next in frequency to the alterations in the spleen.

A few other observers have reported similar cases. In cases in which the secreting apparatus is mostly destroyed, the secretion of the pancreatic juice must be impossible. The disturbed function of this organ must have a disastrous effect upon the nourishment of the child, and is probably a potent cause of the gastro-intestinal disturbances so common in hereditary syphilis.

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[1] Cornil on Syphilis, translated by Simes and White, p. 410.

[2] Medico-Chirurgical Society, March 26, 1867.

[3] British Medical Journal, January 20, 1877.

Larynx, etc.—The pharynx, larynx, trachea, and neighboring parts may be, in early inherited disease, the seat of ulcerations resulting in extensive loss of substance, followed by correspondingly extensive cicatrization and stenosis. The destruction of the palate so characteristic of syphilis is undoubtedly sometimes the result of hereditary disease, but more often in its later forms. In an article on congenital syphilis in the throat, Dr. J. N. Mackenzie, of Baltimore, finds that, of thirty cases of deep ulceration of the palate, pharynx, and naso-pharynx, fourteen occurred within the first year and ten within the first six months of life, the remainder occurring at periods more or less advanced towards puberty. The occurrence of so large a proportion so early seems incredible. The results of the destruction of palate and naso-pharynx will be referred to further under the head of late hereditary syphilis.

Thymus.—Disease of the thymus has attracted no little attention as a sign of hereditary syphilis. Abscess of the thymus occurs in some cases, but is not a constant nor very reliable symptom. The secretion of the thymus closely resembles pus, and cannot always be distinguished from it.

Heart.—The heart has been found to contain gummata, and Dr. Coupland has described a specimen in which the muscular walls were thickened and hardened and showed under the microscope an almost universal infiltration of small round cells among the muscular fibres. In the same case the kidneys, although normal to the eye, were seen to be undergoing similar changes, and their substance was unnaturally firm.[1]

Lungs.—In the syphilitic fœtus born before term, and in syphilitic children who live a few days, there are found, at the autopsy, in the lungs nodules or small tumors, usually superficial, sometimes deep, hard, isolated or in groups, pink, gray, or red in color, with scattered whitish or yellowish points, varying in size from a pea to a small walnut. On section they are spherical or lobular, and present the same small whitish points throughout. These nodules are scarcely prominent, and represent simply a part of the lung more or less considerable in a state of special lobular hepatization. At other times an entire lobe may be involved. The dense altered part usually sinks immediately when placed in water; it is colorless, gray, or white, both on its surface and on section. The pleura is always affected,—thickened and inflamed.[2] This condition is called by Virchow *pneumonia alba*, white hepatization. The lungs are voluminous, and bear the impress of the ribs.

Kidneys.—At the discussion upon renal syphilis before the Clinical Society of London in 1880, Dr. Coupland reported two cases of hereditary syphilis,—the first a girl of thirteen with marked hereditary disease, the second a girl of eighteen in whom the diagnosis of syphilis was fully established. The kidneys in both cases presented the lesions of parenchymatous nephritis. At the same meeting, Barlow expressed the opinion that patients with congenital syphilis are very susceptible to or are predisposed to nephritis, while Mahomed believed that cases of nephritis due to syphilis are primarily cases of amyloid degeneration.

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[1] Eustace Smith, Diseases of Children, p. 205.

[2] Cornil on Syphilis, p. 404.

A further contribution to our knowledge has been made by Parrot. He found the kidneys on section studded with numerous small tumors, varying in size from a pin's head to a cherry-stone. The smallest were white, and the larger were yellow in the periphery and reddish at their centre. The lesion consisted primarily of a circumscribed or diffuse infiltration of round embryonic cells with others of fusiform shape into the connective-tissue framework, followed secondarily by compression or destruction of the tubules and colloid degeneration of their epithelium. In the first stage the organs are enlarged : in the second, general atrophy sets in, and they are finally much reduced in size.

Children affected with hereditary syphilis may die early with symptoms which may be referred to the kidneys, or they may recover in spite of renal lesions.

Testicle.—The lesions of the testicle in hereditary syphilis that are appreciable during life are perhaps not very common, but the affection often exists in a state so little advanced that it needs the microscope to discover it. Its alterations have been studied by Cornil and Coyne, Parrot and Hutinel, Lewin, Taylor, and others. The testicles are slightly enlarged and harder than normal; the epididymis is normal. Both testicles are similarly changed and uniformly altered, or the interstitial orchitis may be unilateral and irregularly distributed. The lesions begin in the connective-tissue framework, and offer a most striking resemblance to the hepatic lesions. The lesion consists in small collections of round embryonal cells resembling lymph-cells, arranged in the connective tissue around the arterioles which come from the tunica albuginea ; or there is only seen a thickening from the new formation of small round cells of the connective tissue of the testicle. The seminal ducts are all surrounded with these cells, and the gland undergoes hypertrophy. The cells within the ducts become granulo-fatty and atrophied. The ducts are also atrophied.[1]

Henoch reports seven cases of syphilitic disease of the testicles in children from three months to two and one-half years of age. Taylor has also reported five cases.

Osseous System.—In still-born infants and in those dying soon after birth, the majority or even all of the long bones are affected. So common are the affections of the bones that it is doubtful if they are ever entirely absent in a fœtus which is really syphilitic.

In the growing infant the epiphysis is joined to the shaft by a layer of cartilage. It is at this cartilaginous layer that growth in the length of the bone takes place, and here syphilitic changes are most often found. The lesion is an osteochondritis, and may be the sole manifestation of the disease or may occur in conjunction with skin and other lesions. The bones most commonly affected are those of the forearm, the leg, the arm, and the thigh. As a rule, several bones are affected symmetrically and simultaneously.

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[1] Cornil on Syphilis, p. 421.

In the living infant the clinical form usually taken is that of a tumor at the junction of the diaphysis and epiphysis at the distal end of the long bones, though any part of the osseous system may be involved. These swellings are difficult to recognize in fat children. The tumors rise abruptly from the bones, being smooth and globular, in some cases forming a ring at the junction of shaft and epiphysis, in others the whole epiphysis is enlarged. In some cases, only a part of the cartilage is affected and the external swelling is correspondingly circumscribed. The lesions appear soon after birth, and may complete their development either slowly or rapidly. The termination varies widely. The swelling may be absorbed under appropriate treatment, or suppuration may take place and the skin break down; the disease may end in the separation and destruction of the epiphysis. The result upon the final growth of the limb varies, of course, with the severity of the local disease. When the morbid process is arrested before the destruction of either cartilage or epiphysis, no deformity results, but the destruction of cartilage of course puts an end to growth at that point, and a more or less shortened and useless limb results. When the disease takes such a course as to separate the epiphysis while the integuments remain sound, the limb becomes useless for a time and appears to be paralyzed, a condition described by Parrot and known as Parrot's disease or pseudosyphilitic infantile paralysis. The joints in immediate connection with these diseased bones are sometimes involved. There may be simple effusion, or, where the bone is destroyed, of course serious trouble to the joint must follow.

Osteochondritis is ordinarily the form of bone-disease in infants. Osteoperiostitis belongs almost exclusively to the later forms of hereditary syphilis as they appear in well-grown children or young adults.

The fingers and toes are also subject to a peculiar form of disease in infancy, of the same character as that occurring in acquired syphilis, known as dactylitis syphilitica. The phalanx involved may be enlarged to two or three times its natural size. One or several fingers or toes may be involved, and sometimes the metacarpal bones are also diseased. The proximal phalanx is most often affected, and the distal phalanx least often. In the early stages the integument is unchanged; later the overlying parts become inflamed and abscesses form. If the case is submitted to early treatment the deformity usually subsides, but untreated the disease may result in permanent deformity and uselessness.

In addition to the purely syphilitic changes already mentioned, local thinning of the bones of the skull, called *craniotabes*, occasionally occurs. In this condition the bone-substance is absorbed, leaving only the integuments and membranes, and softened spots, nearly circular in form and about half an inch, more or less, in diameter, may be recognized by the finger, during life. Until lately craniotabes was considered to be exclusively a symptom of rickets. It is found especially in the occiput, and is thought to be the consequence of compression of the bone between the brain within and the pillow without. It is present in rickets where no trace of syphilis can be discovered, but it seems to be most common in cases where there is also a distinct syphilitic taint. Of one hundred cases of craniotabes collected by Drs. Barlow and Lees,[1] in forty-seven there was satisfactory proof of syphilis, in forty there was evidence of syphilis that fell short of demonstration, in twelve only was there no evidence of syphilis to be detected.

Clinical History.—A disease which pervades the whole economy and may manifest itself in any of its parts or in any number of parts permits of an infinite variety of combinations and a corresponding difficulty in description. Such is practically the condition that confronts us in the clinical study of syphilis. There are, however, many symptoms which are nearly constant.

Earliest Manifestations.—If the disease is manifest in the child at birth, the symptoms are usually severe. The child is emaciated. He snuffles, and cries hoarsely. An eruption of bullæ appears, situated principally on the palms, soles, wrists, and ankles, and often confined to the extremities. These bullæ are filled—or, rather, partially filled—with a semi-purulent fluid. On the palms and soles particularly, they burst, leaving angry-looking sores, which remind one somewhat, by their situation and general appearance, of the palmar and plantar syphilides of adult syphilis. The cachectic look and general feebleness of these children show them to be profoundly affected. The lips are cracked and ulcerated, and crusts form at the corners of the mouth and openings of the nostrils. The liver and spleen are manifestly enlarged, and the imperfectly-performed digestion, as shown by continued loss of flesh and unhealthy stools, awakens the suspicion that other abdominal viscera are also involved. These cases usually prove fatal in a few days or weeks, often in a few hours.

It must not be considered that all cases that show the disease at birth answer this description. The symptoms are not invariably so marked, nor do all these cases prove fatal.

Of syphilitic children who are brought living into the world, a very large proportion show no signs of the disease at birth. The child usually has all the appearances of health. But, though most of these children are born apparently healthy, presenting no symptom by which the most practised eye can detect the disease, some of them, without showing signs of syphilis, show that they are not perfectly healthy. The skin is unnaturally pale, or dull and muddy-looking.

The disease shows itself almost invariably within the first three months, and usually within the first two. Kassowitz, out of four hundred cases, found that the disease manifested itself in the first month in fifty-three per cent., in the second month in thirty-two per cent., and in the third month in the remaining fifteen per cent. Out of fifty-three cases in the Farringdon Dispensary, Dr. B. W. Dunn[1] found that seventeen cases first manifested the disease in the first month, twenty-one in the second month, ten in the third month, two in the fourth month, one in the fifth month, and one in the sixth month. Miller, of Moscow,[2] from a study of a thousand cases, found the first appearance of symptoms in the first month in sixty-four per cent. and in the second month in twenty-two per cent. The first symptoms occurred in the third week of life in twenty-four per cent.

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[1] Lees (D. B.) and Barlow (T.), Med. Times and Gaz., London, 1880, ii. 611.

Sometimes the outbreak is determined by a febrile disease, such as one of the exanthemata. Thus, the rash of measles may subside leaving the syphilitic eruption in its place.

Wakefulness.—One of the earliest symptoms, which is little noticed by authors, but is seldom absent, is obstinate wakefulness at night. According to Eustace Smith,[3] the child when put to bed is uneasy and wakeful; he cries almost unceasingly, and cannot be pacified. During the day he is more quiet, but every night there is a repetition of the same disturbance, and his uncontrollable complaints are a source of perplexity to his attendants. The crying is possibly excited by nocturnal pains in the bones, similar to those affecting adults. The sleeplessness often continues after the appearance of other symptoms, but it ought soon to subside under the influence of mercurials. In an infant born perfectly healthy in appearance (of a mother syphilitic about a year), and showing a pemphigoid eruption during the fourth week, which was the reason for medical consultation, the mother complained, in giving its history, that the child would not sleep at night, even from the day of its birth.

I have recently seen the second child of a syphilitic family in which the first child showed the obstinate wakefulness above noted. The second child, which has shown but slight signs of the disease up to the age of six months, is not wakeful as was the first child, but often starts screaming out of a sound sleep, according to the mother's story.

Snuffles.—Nasal catarrh attacks a very large proportion of syphilitic infants, and gives rise to a most characteristic symptom. It appears early, often, if not always, preceding the eruption. It shows itself at the beginning by difficult and noisy respiration. The mucous membrane of the nose becomes swollen, and partially closes the nasal passages. A nasal discharge appears and increases, still further occludes the passage, and acts as a valve with each respiration, which becomes noisy. The symptom thus caused is known as the *snuffles*. The discharge from the nostril is sero-purulent in character at first, and often streaked with blood. In severe cases this discharge runs down over the upper lip, which becomes reddened and excoriated. The difficulty of respiration increases; the discharge becomes purulent and dries into crusts, which may entirely close the nostrils and oblige the child to breathe through the mouth. In such cases nursing becomes difficult and often impossible; the child, obliged to relinquish the breast every moment to get breath, is fed very imperfectly, and sometimes weans himself.

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[1] Brit. Med. Journal, 1865, ii. 600.

[2] Jahrb. f. Kinderh., xxvii., Hft. iv. S. 359, and Viertelj. f. Dermat., N. S., 1888, Hft. iv. S. 649.

[3] Wasting Diseases of Children, 2d American ed., p. 184.

The nasal symptoms may be very persistent, continuing months after other symptoms have vanished. In some cases the snuffling is not very noticeable so long as the child lies quiet and breathes through the mouth, but the difficulty becomes at once apparent if he is disturbed, and even more marked when he takes the breast. He is then obliged to breathe through the nose, and each respiration is accompanied by a snuffle.

The discharge itself is attributed by Diday and others to mucous patches on the Schneiderian membrane. Usually the inflammation in the nasal fossæ involves only the mucous membrane, and is rarely propagated to the periosteum, the cartilages, and the bones. In the severest cases the ulceration may, after a time, perforate the septum nasi or lay bare the nasal bones, which become necrosed in consequence of the exposure. Fragments of these bones are sometimes found in the crusts thrown off.

In some cases there follows a depression at the root of the nose. Trousseau considers this deformity very frequent. There is, however, a deformity—either a lack of development in the nasal bones or a preternatural widening—which is common in syphilitic infants, but it is not certain that it is not a congenital deformity rather than a result of the local disease which causes snuffles. Absolute destruction of the bony framework at this early stage must be rare.

Coryza may be the only symptom of syphilis in the infant, though its occurrence without other signs of the disease must be far from common. The possibility that it may occur alone makes the diagnosis difficult sometimes. Syphilitic snuffles may be confounded with a simple cold—an exceedingly common error with the family—or with the nasal discharge from diphtheria, perhaps with nasal or retro-pharyngeal polypi. Simple coryza is extremely frequent, even at a very early age. The discharge is less sticky and less inclined to form concretions than that of syphilis. At the end of eight or ten days it tends to disappear, while that of syphilis, if left untreated, persists and increases. The nasal discharge of diphtheria might be difficult to distinguish if diphtheritic patches did not exist in the throat. The diphtheritic discharge, which presents nothing peculiar at first, after twenty-four to forty-eight hours is streaked with blood; and the nasal mucous membrane may be covered with false membrane. The rapid march of diphtheria will not permit any doubt to be of long continuance.

The cry of the syphilitic infant is a most noticeable feature in the severe cases. It is at once hoarse and high-pitched. Its peculiar quality is due, without doubt, to the existence on the vocal cords of lesions similar to those that cause the nasal symptoms.

Skin and Mucous Membranes.—Ordinarily the symptoms already mentioned are but the forerunners of cutaneous manifestations.

The skin presents a series of eruptions which closely resemble those of acquired syphilis, but are modified by the character of the infant's skin and by the manner of its life. The nates and those portions of the body about the pelvis which are moistened and smeared over many times a day are particularly liable to be the seat of eruptions, or a general eruption will flourish and take on more marked characteristics in that region. As already mentioned, the eruption of bullæ occurs about the hands and feet in cases of unusual gravity. In cases in which the eruption is delayed till a later period, the usual eruption is an erythema, which consists of round or roundish pink spots which disappear on pressure at first. Soon the spots grow darker, assume the dull-red coppery hue, and no longer disappear on pressure. Not infrequently a papular syphilide may be the first manifestation on the skin. The syphilides, as the eruptions of syphilis are called, are separately described in a special article on the subject.

The skin hangs in folds on those cachectic subjects in whom emaciation is marked, but wasting is not a prominent symptom in many of the cases. The skin has a pale, sallow, yellowish, or earthy hue which is often said to be characteristic; but diagnosis from the hue of the skin alone is an exceedingly delicate matter.

The manifestations on the lips and buccal mucous membranes are of capital importance. On the lips fissures, known as rhagades, are exceedingly frequent. Their number and depth are very variable. On the upper lip they occur especially on either side of the median lobule, where they are manifestly an exaggeration of an anatomical disposition. On the lower lip the fissure is often a single one in the median line. In addition, the whole surface of the lips may be covered with ulcerations and excoriations. At the angles of the mouth also, flat papules on the muco-cutaneous portions, condylomata, and ulcerations occur, which may be covered with crusts or be superficially or deeply ulcerated. A peculiar appearance is occasionally imparted to the mouth by muco-cutaneous ulcerations at the commissures, which look as though the mouth had been lengthened by a slit at the angles.

Tongue, gums, and fauces may also be more or less ulcerated. At first the manifestations of disease in the mouth consist of slightly-elevated, well-defined portions of mucous membrane with whitish surfaces, like the corresponding manifestations in the adult. The whitish epithelium is often cast off, leaving a smooth, often depressed surface, which may ulcerate. In the severer cases these patches lose their regular outline, coalesce, and form ulcerated surfaces of considerable extent.

The secretion from these ulcerations is abundant and highly infectious.

It is the source of inoculation in most of the very frequent cases in which heredito-syphilitic infants spread the disease.

Adenopathy.—Enlargement of the glands seems to be a less marked characteristic of hereditary syphilis than of acquired. Lymphatic ganglions are said by Parrot to be less developed and less sensitive in very young children than they are a little later. When they are enlarged in infantile syphilis, Parrot thinks it is consecutive to cutaneous lesions; but I have certainly felt the cervical glands when no cutaneous lesions were present to account for them. In order of frequency they are the inguinal, axillary, and cervico-maxillary. Their characteristics are the same as in adult syphilis: they are multiple, non-inflammatory, perfectly distinct and movable in their cellular atmosphere. They are seldom recognized except by touch, though occasionally they project sufficiently to be noticeable to sight. It is evident that the general enlargement of glands is of much less diagnostic value than in acquired syphilis.

It is only lately that I have paid much attention to the glands. A few observations lead me to think that enlarged glands from syphilis are unusual in very young infants, but that they are very common in children who have reached the age of a few months.

On post-mortem examination the bronchial ganglia of a syphilitic child five months old were found infiltrated by Hutchinson, and the glands of the omentum and mesentery have also been found enlarged.

Alopecia.—The same loss of hair occurs in the inherited as in the acquired form. It may happen from the occurrence in the scalp of dermal lesions, but there is a loss of hair due probably to the adynamic influence of syphilis, which is more or less severe in different cases. I have recently heard a mother speak of her first syphilitic but sixth child as the only "bald-head" in the family. As the baby's head was covered by a growth of fine but short hair, I asked her reason for so designating the child, and received the following explanation. The last child, like the others, was born with an abundant growth of hair, but, while the healthy children had retained their original growth, in the syphilitic child the long and dark hair present at birth had gradually fallen and been replaced by the shorter new growth, so that by comparison the child seemed bald.

Occasionally the eyebrows and eyelashes are lost.

Barlow, in a short article on alopecia in congenital syphilis, says that he has seen several syphilitic children in whom alopecia has occurred. In some of them, all he could say was that in a given patch the hair was very much thinned, in others the loss of hair has been as marked as in alopecia areata. He thinks that in many of the cases which he has seen the loss of hair has been preceded by desquamation (sometimes very slight) of the scalp in the region which has subsequently become bare.

He believes the eyebrows are the most conclusive spots. "If in a child from two to three months old one or both eyebrows be bare, it ought always to raise the suspicion of congenital syphilis. The occipital region has in some cases been affected, and with it there has been a moderate enlargement of the occipital glands."

There are two provisos to be borne in mind: 1st, that in rickety children with much head-sweating and muscular weakness the occiput very often becomes almost bare of hair; and, 2d, that in a young baby the hair presents a deep bay where the hair is deficient in each fronto-temporal region; unless the alopecia is very marked on one side, it is hardly characteristic.

Onychia.—The nails are quite frequently involved in hereditary syphilis,—more frequently than in adult syphilis. The disease occurs in two forms. In the first form a papule or pustule occurs on the skin at the side of the nail. It ulcerates and reaches and extends along the side of the nail. It may involve the matrix and cause the loss of the nail. The thick and everted edges of the ulcer, its sloughy base, and the sanious discharge are more or less characteristic, and are accompanied by a general and painful enlargement of the distal phalanx. The second form of onychia is a later manifestation. It begins as a swelling at the base or side of the nail, which becomes thickened, fissured, and brittle, with more or less deformity of the phalanx.

Dentition.—Syphilis sometimes brings about a retardation more or less marked in dentition. Under its influence the infant cuts its first teeth only in the tenth, twelfth, fourteenth, or fifteenth month, and sometimes even later. This retardation in dental evolution when it occurs is usually general,—that is, it involves equally the whole dental system. It may, however, localize itself upon a single group of teeth, as, for instance, the incisors. A like retardation may affect the appearance of the permanent teeth. The primary teeth are especially prone to premature decay. The teeth of the second dentition undergo most important changes, and are believed to present deformities which are pathognomonic and which will be mentioned later. An abnormally early appearance of the teeth is frequently associated with signs of syphilis, and is followed by an early decay. Unfortunately, the changes in the deciduous teeth are not sufficiently characteristic to be of diagnostic value.

Iritis in Infants.—Mr. Hutchinson has observed twenty-three cases of iritis in syphilitic infants. The average age at the commencement of the iritis was five months and a half. The oldest was sixteen months at the time of the outbreak, the youngest six weeks. Both eyes were attacked in eleven cases. In fifteen cases the effusion of lymph may be said to have been copious. The cornea was implicated in a few cases. In seven cases the cure was complete. In twelve cases the pupil was permanently occluded. Iritis must be considered as one of the rarest of the symptoms of hereditary syphilis, but Mr. Hutchinson thinks that it often escapes notice on account of the very slight symptoms which usually attend it. Infants suffering from iritis almost always show some of the well-recognized symptoms of hereditary taint. Mercurial treatment is most efficient in averting the blindness that results when patients are left untreated.

Digestive Troubles.—When gastro-intestinal disorders appear in a syphilitic child, it is necessary to inquire what part syphilitic lesions in the liver, the spleen, the pancreas, and perhaps even in the stomach and intestines, play in their genesis. The symptoms begin in an insidious manner, differing not at all from symptoms observed in non-syphilitic children,—viz., regurgitation, vomiting, and diarrhœa. The symptoms persist in spite of treatment, change of nurses, and the most careful hygienic and dietetic care. The child emaciates rapidly, until fat and muscles seem entirely absorbed. In others a mild diarrhœa will persist, although the child is doing in other respects remarkably well. It is probable that erythematous changes similar to those seen in mouth and pharynx exist lower down in the digestive tract to account for some of these symptoms: actual structural changes have been found. Förster[1] has described a fibroid degeneration of Peyer's patches in a syphilitic infant who died six days after birth; and his observations have been confirmed by others.

Syphilis Hæmorrhagica Neonatorum.—Of this somewhat rare affection Bumstead and Taylor reported in 1883 that sixteen cases existed in literature, and added two more from their own experience. Clinically the hemorrhages vary in extent and severity. In some cases there is merely a limited subcutaneous effusion, in others the hemorrhage takes place into the substance of, or on the surface of, mucous membranes. Some of the fatal cases of umbilical hemorrhage belong in this category. As the hemorrhages occur only in very young children,—seldom later than the first month,—it is often difficult to be certain of their cause.

Dr. Uracek[2] has reported a series of peculiar hemorrhages in different internal organs. Out of one hundred and thirty-two deaths among infants of syphilitic mothers, slight hemorrhages were distinguished in forty-four cases. There were only nineteen cases, however, in which the positive diagnosis of syphilis could be made. Of these children, eighteen came into the world alive, but none lived very long. Ten died within a quarter of an hour.

An interesting case recently reported by Dr. J. Harris Jones[3] illustrates the difficulty of a positive diagnosis. It occurred in a family in which the father was known to be syphilitic, and in which other children had died in infancy, the last one from syphilis. Dr. Jones was summoned to see the child in question—a male—on the fourth day after birth, as the child had bled slightly from the umbilicus. There were several large, unmistakably purpuric spots over the chest, abdomen, and armpits. There had been some bleeding from the nose, and the nurse had observed a little blood on the napkins that morning. In spite of treatment, the child continued to grow worse. Fresh purpuric spots appeared, and the epistaxis and melæna increased in severity. The urine on a few occasions was bloody. The child died on the eleventh day. The doctor could assign no cause for purpura in this case other than syphilis.

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[1] Würzb. Med. Zeitschr., vol. iv. part i., 1863; also reprint.

[2] Deutsch. Med. Zeitung, No. 82, 1886.

[3] Brit. Med. Jour., November 12, 1887.

The hemorrhagic cases have usually occurred in families more recently syphilitic than was apparently the fact in this one.

Contagion.—The contagiousness of the lesions of a congenital syphilitic infant is undoubted. The possibility of inoculation from an infant was denied by Hunter, but the observations on which the non-contagiousness was affirmed are now known to have been erroneous. The other extreme has been taken by certain writers, to the effect that an extreme virulence was seated in the hereditary disease. The truth seems to be that it is no more contagious than acquired syphilis, but that the freedom with which an infant is handled and the frequency with which contagious lesions are found in its mouth make it extremely easy for inoculation to take place. A syphilitic infant is a source of danger to the non-syphilitic members of its family, and numerous cases are seen in which the baby has infected its grandparents, its nurses, and other infants. As a matter of course, inoculation from a syphilitic infant gives rise to a chancre in the infected person.

Diagnosis.—The diagnosis of a case of congenital syphilis depends in most cases largely upon the eruption, and upon the presence of certain symptoms already described. When these are fully developed, the diagnosis is comparatively easy; when the eruption has passed away and only doubtful signs are present, the diagnosis is often exceedingly difficult.

In estimating the history of a child suspected of syphilis, absence of history of a rash cannot be considered decisive evidence against a diagnosis of syphilis. A true syphilitic rash is at times so slight in extent and mild in character as to attract no attention. Special weight in favor of syphilis should be given to a history of a rash on palms and soles.

Chronic snuffling is one of the most reliable signs. Any child may snuffle for a time, but if it continues to do so for several months, especially if the snuffling commence soon after birth and if it be at times accompanied by a blood-stained discharge, it is highly suspicious. A child may snuffle when disturbed who shows no sign of it when quiet.

Collapse of the bridge of the nose is a valuable sign when marked. It varies in amount from a condition approaching flatness to one so slight that its existence is questionable.

Enlargement of the spleen in the early months of life justifies a strong suspicion.

Rapid improvement under the free use of mercury, especially when non-mercurial treatment has been unsuccessful, is most valuable testimony to the syphilitic character of the child's ailment, though it alone should not be considered a complete demonstration.

A suspicion of syphilis may be entertained with regard to children who have been brought up at the breast and have not suffered from any digestive trouble, but yet fall into a condition of marasmus. Enlargement of the spleen strengthens such a suspicion, and it approaches certainty if improvement follows the use of mercury.

Prognosis.—The condition of the parents is an important factor in estimating the future of the child. Generally speaking, the earlier in the disease of the parents the child is born, the more likely is that child to die. It is but repeating the same idea in other words to say that the liability to a fatal termination decreases with each subsequent pregnancy. This is not an invariable rule, however.

When both parents are affected, the disease is more likely to be severe than when only one parent is diseased. When the parents have been thoroughly treated, the prognosis is much better for the child.

Prognosis is almost invariably fatal in bottle-fed infants in asylums; it is somewhat better in well-to-do private families.

The severity of nasal symptoms is an important element in the child's welfare. If they are of such a character as to interfere seriously with nutrition, the outlook is less favorable than when the child is able to take its food without hinderance.

The degree of splenic enlargement may be taken as an index of the severity of the disease. The majority of cases of great enlargement of the spleen die.

The prognosis is always a grave one, but it becomes less serious the later the appearance of active symptoms.

When the infant survives, he may apparently throw off all trace of the disease and grow up a strong healthy adult. But when the symptoms have been severe, more or less permanent impression is produced upon the system. The patient is liable to outbreaks of various sorts, the characters of which are more fully discussed in the following pages.

Treatment.—The nutrition of the syphilitic infant is of the first importance. With a peculiar liability to digestive troubles, it is extremely desirable that it shall be nursed; and with the strong probability that it will infect a healthy wet-nurse, the duty of suckling her infant devolves most strongly upon the mother. Even if the mother is manifestly syphilitic, in the majority of cases she will be yet capable of nursing her infant. If her disease is severe her milk may not be of the best quality and may be deficient in quantity, but even under such circumstances it is ordinarily better for the child to allow it to nurse so far as possible, the deficiency being made up by feeding it artificially as many times a day as may be requisite, the mother being subjected to such treatment as her condition requires.

Eustace Smith[1] quotes from Vernois and Becquerel[2] an analysis of the milk of nine women with well-marked constitutional syphilis, which shows that the water and salts were increased while the casein and butter were diminished. Although the quality of such milk is indeed poor, it is better that the child should be suckled so far as possible, rather than trust entirely to artificial feeding. It is also highly probable, as Eustace Smith points out, that where the disease assumes a milder form the milk does not depart so greatly from the normal standard as in the well-marked cases in which the analysis was made.

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[1] Wasting Diseases of Children, p. 151.

[2] Du Lait chez la Femme, par MM. Vernois et Becquerel, 8vo, Paris, 1853.

In those cases in which the mother is apparently healthy though her child is syphilitic, the child should continue to be suckled by its mother. There is practically no reason to fear that the child will injure its mother in accordance with the facts known as Colles's law.

The employment of a healthy wet-nurse for a syphilitic child or for one suspected of syphilis is not justifiable. The syphilitic nursling is almost sure to inoculate its nurse, who in turn is extremely liable to give the disease to her own baby if she is still nursing it and to other members of the family. The question sometimes arises whether the doctor may sanction the employment of a healthy nurse for a syphilitic child if the nurse herself knows the danger to which she will be exposed and is willing to undergo the danger—a danger which, certainly without precaution, amounts almost to a certainty—of taking the disease. Of course there are cases where the parents are ready to do anything to raise the child, and where the nurse from some motive, pecuniary or otherwise, is willing to run all risks. Cases of this sort present special features which must influence the doctor in his action; but in giving even a quasi-sanction to such a sacrifice on the part of the nurse he must make sure that she knows the risk she is running. The doctor alone can comprehend the possible future such a sacrifice may entail, and he must be in some measure the guardian of the woman and of the public. He ought to make sure that the compensation the nurse receives is in proportion to the risk run. He ought also to make sure that the nurse's husband and children, and through them the community, are protected. If after all the nurse accepts the care of the syphilitic infant, pains must be taken to avoid inoculation. The baby should be properly medicated in accordance with the views that follow. Its mouth and lips should be kept in as healthy a condition as possible. The best protection for the nurse is the use of an artificial nipple. Women who have had syphilis and recovered are not exposed to inoculation, and if a wet-nurse who answers that description can be found she is a proper person to nurse the syphilitic infant. Women who have borne syphilitic infants while remaining themselves apparently healthy are also eligible.

From what has been said, it follows that artificial feeding must often be resorted to. It will require the best judgment of the attending physician, who must expect to lose a large number of artificially-fed syphilitics; but in principle it is not different from feeding other babies, and needs no further comment here.

The medicinal treatment of the syphilitic infant is conducted on the same principles as that of the adult acquired disease. The same drugs exercise like powers on the adult disease and the infantile disease, due regard being paid to the appropriate dose and preparation.

The favorable effect of thorough treatment of the parents upon their unborn offspring must be remembered. If the opportunity is afforded, the pregnant syphilitic woman ought to be thoroughly treated, for the sake of her unborn infant as well as for her own.

When syphilis is evident in the infant, it is necessary to interfere immediately and have recourse to mercury. Young infants support the drug well, and the practitioner should feel confidence in its curative properties, but it is necessary to take account of the age of the child for the dose, and of its general condition for the mode of administration.

As a rule, internal medication is perfectly simple, and no preparation is better than mercury with chalk. Of this half a grain or even a grain may be given night and morning, the dose being increased by a fraction of a grain every few days until the infant is taking two grains twice a day. To prevent any irritating effect upon the alimentary canal, Eustace Smith advises the addition of a grain of carbonate of potassium or a few grains of prepared chalk to each dose. If, in spite of this addition, derangement of the stomach and bowels be excited, it will be better to have recourse to inunction.

Calomel is also extensively used, and is the form preferred by Jacobi. It is sometimes effectual in calming irritability of the stomach when excited by other forms of mercury or when it exists from independent causes. It may be given in doses of one-twentieth to one-sixth of a grain three times a day. Its use is sometimes attended with diarrhœa, but it can ordinarily be given for months at a time. If diarrhœa should be excited without fault in the alimentation, one-twentieth to one-twelfth of a grain of Dover's powder might be added to each dose; but ordinarily the addition of an opiate is to be avoided. The bichloride of mercury in solution is an extremely convenient form for administration. Keyes[1] recommends a solution of half a grain in six ounces of water. Each teaspoonful contains one-ninety-sixth of a grain. This solution has absolutely no taste, and the child will believe it to be water. It may be given with milk, when its presence will be unsuspected. Of this solution a teaspoonful should be given every three or four hours for prolonged treatment, the interval being shorter when it is desired to produce a rapid result.

Parrot believed the liquor of Van Swieten to be the only preparation that can be properly administered to infants.

One of the most satisfactory methods of treatment is inunction by means of mercurial ointment diluted with an equal quantity of petrolatum. With this ointment a piece of cloth large enough to cover in great measure the child's abdomen is thickly spread and placed under the flannel bandage. It is renewed daily, and its position may be shifted from front to back or side as often as any sign of irritation appears, or regularly so as to forestall any irritation. The movements of the child serve to keep up a slight friction, which is sufficient to introduce the mercurial into the economy. The application of the ointment by actually rubbing the skin with a ball of cotton or a swab covered with the mercurial is sometimes advised, but is a less satisfactory method than the constant application.

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[1] Venereal Diseases, p. 247.

Baths of corrosive sublimate solutions are occasionally resorted to, but are probably less reliable than inunction.

It is an important question, and one often asked, whether mercury is really curative or whether it simply masks the symptoms.

So far as its control over symptoms is concerned, it is most marked. It is not too much to say that its use often rescues infants from impending death. If any drug can be said to be curative in any disease, mercury is curative in infantile syphilis. If it is not an antidote to the poison of syphilis in all cases, it certainly comes very near it. When we consider the marked pathological changes which syphilis causes in the infantile economy, it is not strange that many cases should prove fatal in spite of the best treatment. If any drug of equal power should be newly discovered for any other disease, it would be hailed as the greatest of blessings.

Iodide of potassium has the same uses as in adults,—that is, it is of special value when the bones or the nervous system are the special objects of attack. There is a tendency on the part of those who see but few cases of congenital syphilis to feel that iodide of potassium is a milder drug, that less risk is run in medication by its use than by the use of mercury, and that the iodides are only less valuable than mercurials. But mercury is so well borne, and its good effects are so marked, that no fear should be felt in giving it in proper doses. Mercury should be regarded as the important curative drug, and the iodides as most important adjuncts, in infantile syphilis. In the later manifestations the *rôle* of the iodides is more important. Though the iodides should be given in moderate doses at first, they may be given in very considerable doses in quite young children. Dr. L. Emmett Holt reports a case of great enlargement of the liver and spleen in which the child, twenty-one months old, took thirty grains of iodide of potassium daily for a long period, with great advantage. Dr. Holt states that after three years of age children will bear almost as much as adults, and at all ages tolerate it exceedingly well, provided only it be given well diluted, preferably in milk.[1]

The iodides may be joined with a mercurial, as in the mixed treatment of adults. The iodide should always be given in solution in water or in milk, and the mercurial may be administered by inunction at the same time; or the two may be combined in one prescription, like the following from Bumstead and Taylor:[2]

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[1] Arch. of Pediatrics, 1888, vol. v. p. 45.

[2] Fifth edition, p. 835.

℞ Hydrarg. biniod., gr. i;		06
Potass. iodid., ʒiv;	15	
Syr. sarsap. comp.,		
Aquæ, āā, ℥ii.	60	
M.		

Of this mixture a child a month old may take five drops three times a day, increasing the dose by a drop every five days.

The syrup of sarsaparilla is very unpalatable to many children, and a more agreeable syrup may be substituted for it, as it is not at all probable that it possesses any great virtue as an antisyphilitic.

Of the iodides, the potassic salt is the most useful. The iodide of sodium ranks next to it. The iodide of iron is often prescribed. Its virtues have been highly extolled by Monti, though he does not advise its use in severe cases. In point of fact, it is nearly if not quite inert as an antisyphilitic.

Treatment ought to be continued for several months after the disappearance of all external manifestations of the disease.

A question that often presents itself at the very outset is as to the propriety of commencing treatment of a babe apparently healthy, but born of syphilitic parents. Parrot would await manifestations, but would commence treatment in the absence of external manifestations when there existed obstinate intestinal affections not due to athrepsia.[1]

Fournier gives more definite rules as to treatment from birth, as follows:

(1) An infant born healthy—in appearance, at least—of a syphilitic father need not be treated. One knows that paternal heredity is much less certain than maternal heredity; consequently, the infant has chances of having escaped the syphilis.

(2) A child born healthy—in appearance, at least—of a mother formerly syphilitic, and who has not shown any accidents of syphilis during her pregnancy, need not be treated, since, if there are chances of its being syphilitic, there are also chances of its not being so.

(3) A child born healthy—in appearance, at least—of a woman recently syphilitic—above all, if she has had venereal accidents in the course of her pregnancy—ought to be treated energetically from its birth, since it is certain, in spite of all appearances to the contrary, that it is syphilitic, and that its latent syphilis may declare itself at any moment and give rise to grave—even fatal—accidents.[2]

Local Treatment.—It is essential to cure the external manifestations, especially those in the vicinity of the mucous outlets which produce a discharge, as soon as possible, because of the highly contagious character of the discharge. Of the local lesions, the most important is the nasal trouble.

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[1] Parrot's term for a chronic state of malnutrition in infants, attributed to faulty development, characterized by progressive emaciation, greenish evacuations, vomiting, etc.

[2] Gazette des Hôpitaux, No. 150, 1887; Ann. de Derm. et de Syph., April 25, 1888, p. 247.

There can be no doubt that the destructive tendencies of the ulceration are aggravated by allowing crusts to accumulate and block up the nostrils. Such crusts should be softened by warm water applied on cloths, by camel's-hair brushes, or by spray, and carefully removed. The inside of the nostril should then be smeared with some mercurial preparation, such as the white-precipitate ointment, or the ointment advised by Diday of two to four grains of calomel to a drachm of lard, or even the mercurial ointment diluted with once or twice its weight of unguentum petrolei.

Mucous patches of the lips may be lightly dusted with calomel or smeared with an ointment which contains it. Mucous patches about the anus and genitals cannot be better treated locally than by dusting them with calomel and covering them with cloths wet with a dilute solution of chloride of sodium. It is often advised to treat such patches with nitrate of silver, but such applications must be seldom necessary. Local applications are secondary to internal medication, but are themselves a valuable means of constitutional treatment when the child presents any considerable extent of raw surface.

Ulcerated spots, whatever may have been the original lesions, may be dressed with some form of mercurial, and usually do better under such an application than under any other. A general preference for a dressing containing mercury does not imply that the general rules that would govern the choice of dressing for other sores should be disregarded. In some of the syphilitic ulcerations, as in the indolent but destructive ulcers about the nails, the local application of mercury is most important.

LATE MANIFESTATIONS OF CONGENITAL SYPHILIS.

The heredito-syphilitic infant ceases usually to show symptoms of his disease before the end of the second year. Apparently, many patients show no further signs of the disease; others go on without manifestations of the disease until they arrive about the age of puberty, or later. With still others the disease manifests itself at irregular intervals, so that they present a history of nearly continuous trouble or of more or less frequent outbreaks. The first two years, then, comprise the early symptoms of hereditary disease, those that correspond with the secondaries of acquired syphilis: the symptoms that come later may be classed as late hereditary disease.

The evidences of late hereditary syphilis may consist in the evidences of previous lesions, in the modifications of growth that take place under the influence of the disease, and in lesions still active at the time when the patient appears for advice.

The evidences of previous trouble may vary very greatly, according to the age of the patient and the character and severity of previous lesions.

It is an interesting question whether the later forms of the disease ever occur without the previous manifestation of the disease during infancy. Such a question is equivalent to the question whether acquired syphilis in the adult ever shows its late forms without any early secondary manifestations. It is certain that late forms of the disease exist in patients in whom we find no evidence of earlier trouble, or, at best, but very questionable evidence; but it is at least probable that the disease never exists without some early characteristic symptoms, though such symptoms may be very ill defined and are perhaps impossible to trace.

The late forms of the hereditary disease assume the same protean aspects as do the later forms of the acquired disease. Probably all the forms of acquired syphilis may appear in the hereditary disease. The same difficulties of diagnosis are presented in late hereditary syphilis as in the more familiar acquired disease, and it is amenable to the same treatment.

I have preferred to treat this portion of the subject under a separate head because it has recently been the object of especial study and valuable additions have been made to our knowledge, because there are yet many open questions connected with it which are not fairly discussed under the head of infantile diseases, and particularly because there exists a great tendency to refer the lesions of hereditary syphilis as they show themselves in childhood to scrofula. The boundary lines between hereditary syphilis and scrofula or tuberculosis, and between syphilis and rickets, are not yet definitely drawn. To present as accurate a picture of late hereditary syphilis as possible is to assist in a diagnosis which is often difficult and sometimes impossible. Many of the cases that present themselves can give no assistance as to their history. They do not remember their own infancy, and any fact likely to reflect upon either parent has been carefully hidden from them. So difficult is it to fix absolutely the character of the disease by actual history that the profession as a whole may be said almost to ignore the possibility of its appearance as syphilis in later years.

As here used, the term *late hereditary syphilis* is intended to apply simply to hereditary syphilis as manifested in childhood and in youth, as distinguished from its manifestations in infancy. The division of the disease into two periods gives rise to some difficulties in description, as manifestations upon certain portions of the body are common to the two periods.

The *time* at which the later symptoms of hereditary syphilis most commonly show themselves is as impossible to fix as the time at which the so-called tertiary symptoms appear in the adult. In some children the symptoms are continuous from infancy through the whole of their miserable lives. In others a very variable number of years passes between the infantile and the later manifestations. Judging from the histories of certain patients whom I have seen later in life, an outbreak of some sort must be not uncommon at the age of four or five years, while, according to the view of Mr. Hutchinson, the age of puberty is a very common period for their appearance. Rabl gives a table of the time of the first appearance of late symptoms in one hundred and twenty-seven patients: twenty-three showed symptoms before the age of eight; from eight to eleven years inclusive, the disease appeared in thirty-seven; thirteen showed the first signs of the disease at twelve, the largest number in any one year.[1]

It is very certain that the disease may follow its victim to the close of adult life, usually in the form of successive outbreaks with intervening periods of comparative health. As an example of various outbreaks at intervals of years, a case of Fournier's can hardly be surpassed. In this case it was established that the father had syphilis before the birth of the child; the mother was also syphilitic, as she showed late lesions during the patient's second year. At the age of three months the patient was treated for an eruption which Trousseau considered syphilitic. At five years he was affected with bony lesions of the two elbows. At seven years he suffered from grave ocular lesions which threatened the loss of sight. At twelve one of his tibiæ was swollen. At fifteen began a new series of accidents, involving the bony structure of the nose, with necrosis and afterwards breaking down of the nasal bones, and extensive ulceration of the palate and pharynx, which resulted in extensive loss of tissue and formation of cicatrices. Still later a new ulceration of the upper lip and the nose appeared, and at the same time a necrosis of the alveolus of the upper jaw. At the age of twenty-eight he was again troubled with a gummy syphilide over the hyperostosis of the tibia which was primarily affected long years before.

There are certain general characteristics which betray the constitutional malady which has followed the child from its birth, or rather from its conception. Fortunately for these unfortunates, they are not invariably present, and perhaps no single one can be looked upon as pathognomonic of the disease; but when they appear in conjunction their value is incontestable.

Many of the subjects of hereditary syphilis are remarkable for the retardation of physical development. As infants they grow slowly and begin to walk late. Later, when growth is accomplished, their figures are slight, often much below ordinary height. This failure to reach full height is often very striking. Young men and women of eighteen or nineteen fail to reach five feet in height.

Certain curious characteristics mark this lack of development. In the boy the testicles remain at fourteen or fifteen like those of a boy of ten. The beard consists of a few downy hairs; the hair fails to appear about the genitals. In the girl the breasts fail to develop, the genital and axillary hairs are slow to appear, and menstruation is delayed until eighteen or nineteen or later. The mental development is correspondingly slow. So these patients always give the impression of being five or six years younger than they really are. This arrest of development Fournier characterizes as *Infantilism.*

The cranial and nasal deformities are in extreme cases very striking. The cranial deformity may assume one of several types, or the different alterations may be combined. The forehead is most frequently the seat of morbid changes, and, in the first place, may be much more prominent than normal. In place of describing a curve from the eyebrows to the roots of the hair, it rises almost straight to an exaggerated height, giving a majestic appearance to an individual whose growth is perhaps otherwise immature. Upon this prominent forehead, as an additional peculiarity, or on a forehead of ordinary size, a prominence may present itself on the two sides of the median line. This prominence is situated at the central portion of each frontal bone, equidistant from the median line, and usually equally developed on each side. These prominences occur at the situation of the frontal eminence, and are but an abnormal development of a natural configuration. In extreme cases they are very prominent, and give to the head a square appearance which is very striking. Another peculiarity, which differs markedly from the exaggerated frontal eminences and which is much less frequent, is an appearance which has been compared to the keel of a ship. Here the forehead presents a prominence upon the median line, following the course of the medio-frontal suture. In comparison with this prominence the lateral portions of the frontal bone appear flattened, and in some cases they are actually flattened, thus giving a shape which suggests that of a boat's keel.

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[1] Ueber Lues congenita tarda, von Dr. J. Rabl, Leipzig und Wien, 1887.

Deformities of other parts of the skull are less observable, and may require the assistance of the sense of touch for their recognition. Similar protuberances to those described upon the frontal bones occur on the parietals. Like those on the frontal, they are slight elevations, circular in contour, convex, and in consequence more prominent in the centre, of bony hardness, and not involving the skin. They give to the head the appearance of increased breadth. With them is sometimes joined a sensible depression of the skull at the sagittal suture, the junction of the two parietals. This is the deformity which Parrot has designated as the natiform skull, from a suggestion of the shape of the nates. The representations of the skull in Parrot's illustrations make the propriety of this comparison much more evident than does any living specimen I have ever seen.

In addition there may be marked asymmetry of the skull, and sometimes the child is hydrocephalic. The deformity from the latter condition is not common as a result of syphilis, as hydrocephalus as a consequence of syphilis is apparently not very frequent, and the large majority of infants so afflicted die before reaching childhood.

The nasal deformity is among the best-recognized and most characteristic of the results of syphilis. It consists of a breaking down of the nose consequent upon the destruction of its bony and cartilaginous framework. The contour varies according to the character of the destruction of the framework. When the bones are chiefly destroyed, the nose falls in at its origin, and as it becomes flattened pulls upon the cartilaginous portion in such a way as to produce an exaggerated deformity of the character described as *retroussé*. When the cartilages present the greater loss of substance, the extremity of the nose sinks down and may partially retreat within the upper and bony portion.

Other deformities, less marked in character, are much more common. Chief among them is an exaggerated flat nose, such as would be produced by the pressure of the flat of the thumb upon the root of the nose on a wax statue, which would cause not merely a depression but also a widening, or the flattening may be more such as would be produced by the pressure of the thumb on the apex. The actual destruction of the nasal framework which causes the characteristic deformity is always the result of positive disease. The slighter deformities, which are apparent enough and still differ little from peculiarities in persons not syphilitic, can seldom be traced to any actual outbreak of disease, but are rather congenital malformations, perhaps a part of that general incomplete development which has been mentioned, and which is frequently seen in infants of a few months who are undoubtedly syphilitic.

Congenital syphilis leaves its mark also upon other portions of the skeleton than the skull, by changes that resemble somewhat the tuberosities formed upon the skull and may be described clinically as swellings of the bones. A segment of the bone is larger than natural, the hypertrophy essentially modifying the configuration. These swellings occur upon the long bones upon either the diaphyses or the epiphyses. They are common upon the upper extremity of the tibia, upon the head of the radius and of the ulna, at the malleoli, and at the lower end of the humerus. Swellings of the same sort affect the shaft of the same bones, and also the clavicle, but the bone most often and most characteristically affected is the tibia. The bones of the hand are subject to the same forms of disease. When the tibia is attacked, there is a marked increase in size of a portion of the shaft, most likely its middle third; this enlargement is chiefly in the anterior portion, often more or less irregular, and the sharp anterior edge is transformed into a flat surface. In consequence of the prominent swelling on the anterior portion, the bone has the appearance of a curvature like the curve of a sabre: in reality there is no curvature, the bowed appearance being confined to the anterior part. This deformity may exist in both tibiæ or may be confined to one.

Genuine curvatures of bones may apparently take place under the influence of syphilis.

Teeth.—The teeth present modifications, as might be expected, as the time during which syphilis is most marked in the infant is an important period in dental development. The primary set are sometimes late in appearing, frail, and easily destroyed, but they possess no peculiarity that can be relied upon for diagnosis. Nothing can be further from the truth than to regard a deformed or irregular set of primary teeth as an evidence of syphilis. The only alterations that are claimed as pathognomonic occur in the two superior middle incisors of the permanent set. The alteration in these teeth was first described by Mr. Hutchinson, and they are at present very properly known as Hutchinsonian teeth. These teeth are dwarfed: they are at once too small and too narrow. They are also sometimes called "peg-shaped;" though that name is hardly descriptive. The lines of their sides if continued would meet in a point, whereas a healthy incisor is as broad at the cutting border as at the root, or even broader. The cutting edge presents a notch with concavity downward. At the bottom of the notch the enamel is wanting and the dentine is bare. This deformity of the incisors does not occur in every case of hereditary syphilis. It may exist in but one of the incisors while the other is perfect. Their presence is due, according to Hutchinson, to local trouble in the gums during the first weeks of life,—that is, to a stomatitis complicated with an alveolar periostitis,—their presence or absence being determined by the existence or non-existence of gingivitis. If the infant escapes stomatitis, the teeth will not be damaged. Fournier explains their presence as a defect of development impressed upon a tooth yet contained in the alveolus.

These teeth when they first pierce the gum do not always show the notch. They may have instead a projecting lobe or a thin edge deprived of enamel, the shape of the notch being indicated by a crescentic line at the lower edge of the enamel. This lobe is soon worn away, leaving the teeth with their characteristic concavity. The teeth of syphilitics are usually soft, so that they wear away easily, and the notch may be effaced at a comparatively early age. As a clinical fact, these dental peculiarities are commonly associated with interstitial keratitis.

Other deformities of the teeth are very common in hereditary syphilis, but their exact value as diagnostic signs is not clearly established. Sometimes certain teeth are remarkably undeveloped in size.

Dental erosions have attracted a great deal of attention of late, particularly in France. These consist of transverse lines in the enamel, sometimes one or more in the same tooth. They exist not merely in front, but surround the whole tooth, while a section shows that all parts of the tooth partake of the imperfect development. These erosions occur in other children than syphilitics, and even in animals, but Fournier believes them to be so common in syphilitics that their presence should awaken suspicion.

Mr. Coleman, the dental surgeon who examined the cases which Hutchinson reported in his first paper on the subject, drew attention to another peculiarity which seems to be quite common. In nearly every one of Mr. Hutchinson's cases there was a deficiency in the superior alveolar arch at the anterior portion, so great in some patients that the upper and lower incisors were decidedly separated when the jaws were closed.

Eye.—There is in heredito-syphilitics a remarkable liability to a peculiar inflammation of the cornea. It falls little short of being pathognomonic of inherited syphilis, but must be carefully diagnosed. It usually begins as a cloudiness of the substance of the cornea, with ciliary congestion and irritability. The clouds increase and coalesce until the whole cornea looks like ground glass. It begins without pain or general reaction, and without special congestion of the conjunctiva. The affection begins in one eye, but usually attacks the other also. Mr. Hutchinson says that it is always in the end symmetrical, although in rare cases the interval between the attacks in the two eyes may extend to several years. At its height interstitial keratitis may temporarily, for a few weeks, almost entirely abolish sight, but it usually resolves and after a long time disappears without leaving a trace behind. Such a fortunate result is not by any means universal, however. Opacities are often left behind, sometimes sufficient to form a positive hinderance to vision, at other times discoverable only by a special examination with oblique illumination. The duration of the disease is very variable. The period of actual blindness may last from two months, as a minimum, to eight or nine months, as a maximum. The total duration of the disease varies from six to eighteen months.

Complications may arise in the shape of iritis, not always easy to recognize at its inception when the pupil is opaque, and deeper troubles,—choroiditis, retinitis, etc. The disease occasionally relapses after advancing well towards recovery.

Interstitial keratitis occurs most frequently in female subjects, and is most common between the ages of ten and fifteen, but also occurs much earlier. Fournier reports a case from Dr. Parinaud in which an infant was born with the trouble. Dr. A. Trousseau, out of forty cases of interstitial keratitis, in which the ages varied from three to twenty-five, found the greatest liability between the ages of seven and eleven.[1]

Iritis occurs as a symptom of late hereditary syphilis. It presents usually the following symptoms, which differ little from those that characterize inflammation of the iris as it sometimes occurs among syphilitic infants. There is usually but moderate injection. It is indolent in character, there is little pain and but slight lachrymation and photophobia, but at the same time there is an abundant exudation.[2]

The deeper structures of the eye are not exempt from syphilitic manifestations.

Fournier suggests that possibly we may find in the future that certain other affections of the eye may be included among the results of hereditary syphilis. Among such cases he mentions zonular cataract, amaurosis from atrophy of the optic nerves, etc.

Ear.—Troubles with hearing are not as frequent in late hereditary syphilis as are the diseases of the eye, but Fournier noted them forty times in a series of two hundred and twelve cases.

As a secondary phenomenon auditory troubles are exceedingly frequent with diseases of the pharynx of syphilitic and of non-syphilitic origin. The frequency of diseases of the pharynx in congenital syphilis would naturally result in a frequent involvement of the ear, but these cases present nothing unusual in symptoms or anatomical lesions. But there are direct effects of syphilis upon the ear of a very different order. Otitis media purulenta occurs in syphilitic infants with some frequency, and persists into childhood; but the most remarkable manifestation of the disease in childhood and adolescence consists in a deafness which establishes itself without lesions capable of explaining it, which quickly attains a high degree of intensity, is usually rebellious to all treatment, and persists indefinitely. This deafness is usually bilateral, either attacking both ears simultaneously or with an interval, sometimes quite long, between the attacks. It comes on quickly, without apparent cause, without fever or any general or local reaction, and without pain or discharge from the ear. The deafness is usually absolute, and is often accompanied by subjective noises. The same symptom occasionally occurs in acquired syphilis, usually towards the latter part of the secondary stage. Mr. Hinton, of Guy's Hospital, calculated that one in twenty of his patients suffered from it, and that it was by far the most frequent cause of non-congenital deaf-mutism, and Sir W. Dalby places it next to scarlet fever as a cause of deaf-mutism. It is less frequent in the experience of other aural surgeons. The pathological process which determines this deafness is not yet determined.

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[1] Annales de Derm. et de Syph., July 25, 1887, p. 442.

[2] Girard-Teulon, Bulletins de la Société de Chirurgie, November 22, 1871.

Mouth, Nose, and Pharynx.—Dr. John N. Mackenzie, of Baltimore, has studied with great care the ravages of syphilis in the mouth, nose, and pharynx, and has summarized his work in a series of propositions which are given, slightly abbreviated, below:[1]

"1. That deep ulceration may invade the palate, pharynx, and naso-pharynx at any period of life from the first week up to the age of puberty. Of thirty cases analyzed with reference to the period of invasion, fourteen occurred within the first year. 2. When the eruption of inherited syphilis is apparently delayed until the latter period, that lesions of the palate and pharynx are found with a peculiar constancy, and often first attract attention to the existence of a diathesis of which they are the sole pathological expression. 3. That females are attacked more frequently than males. Thus, out of sixty-nine cases of pharyngeal ulceration, forty-one occurred in the former sex. 4. That ulceration may occur in any situation, but its most frequent seat is the palate. 5. That when situated at the posterior portion of the hard palate the tendency is to involve the soft palate and velum, and thence to invade the naso-pharynx and nose; while situated more anteriorly it seeks a more direct pathway to the latter, which is established by perforation of the bone. 6. That the next most common seats of ulceration, in order of frequency, are the fauces, naso-pharynx, posterior pharyngeal wall, nasal fossa and septum, tongue, and gums. 7. That ulceration, especially that of the palate, shows a disposition to centrality of position, together with a special tendency to caries and necrosis of the bone.

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[1] Congenital Syphilis of the Throat: based on the Study of One Hundred and Fifty Cases, American Journal of the Medical Sciences, N. S., clx. 321.

8. That the tendency to necrosis exists at all periods of life, but especially in early youth, when it is more destructive and less amenable to treatment. 9. That while deep pharyngeal ulceration generally precedes or coexists with similar affections of the larynx, the latter occurs too without evidence of pre-existing pharyngeal lesions. 10. That laryngeal ulceration does not commonly follow the pharyngeal destruction of so-called latent syphilis; those palato-pharyngeal ulcerations found in tardy congenital syphilis tend rather to involve the nasal pharynx and nose. 11. That simultaneous or consecutive ulceration of the palate, pharynx, and nose seems to be characteristic of syphilis, or at least occurs more frequently in this than in any other disease."

Bones.—Among active manifestations in late hereditary syphilis disease of the bones is very common. It occurred eighty-two times in Fournier's two hundred and twelve cases. These lesions may show themselves at any time from earliest childhood to adult age, but are unusual before the age of five years. Fournier describes them under three forms,—osteo-periostitis, gummy osteo-periostitis, and gummy osteo-myelitis.

The osteo-periostitis of the heredito-syphilitic differs in nothing in gross characteristics from the same lesion of non-specific origin, but it possesses many traits which give it a character of its own, if they do not fully differentiate it. It has a special predilection for the long bones, and most frequently of all attacks the tibia. Next in frequency come the ulna, the radius, and the humerus. It is usually, or at least very often, multiple, attacking more than one bone, and when multiple is usually symmetrical,—*i.e.*, when it attacks one tibia it is exceedingly probable that it will attack the other tibia also. The periostitis is often accompanied by the deposit of bone, which produces considerable hyperostoses and consequent change in the form of the affected bone. The osteocopic pains with nocturnal exacerbations which are familiar in acquired syphilis are repeated in hereditary. Fournier says the pain often precedes the appearance of periosteal swelling.

Joints.—It is not possible at the present moment to write a proper description of the effects of late congenital syphilis upon the joints. At the same time it is quite evident that the diathesis is at the bottom of many of these affections, particularly those of the knee. It must be said, however, that in a large number of cases in which the joint appears at first sight to be seriously involved the trouble is really situated in the epiphyses of the articulating bones instead of in the joint-structures proper. Simple pain in the knee has occurred, in cases under my own observation, without swelling, effusion, or tenderness. Fournier describes similar cases.

Dr. Clutton has described a form of hydrarthrosis of the knee[1] which he has observed eleven times in children from eight to fifteen. In these cases the effusion was entirely independent of all bony affection; it affected the two knees alike, though in some cases there was an interval of some months before the second knee showed signs of synovitis; it was insidious in its beginning and evolved itself in a chronic manner; it was more amenable to antisyphilitic than to other treatment. The bones in the vicinity were not enlarged. The children in whom it occurred presented undoubted signs of hereditary syphilis, but no other joints were affected. Hydrarthrosis as an accompaniment of bony lesions in the immediate vicinity is not infrequent.

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[1] Symmetrical Synovitis of the Knee in Hereditary Syphilis, Lancet, February 27, 1886, p. 391.

Pseudo-white-swellings are also described, which really are due to massive hyperostoses of the epiphyses and a slight thickening of the periarticular tissues, and except in gross appearance have no relationship with the articular affection which they simulate. These pseudo-white-swellings occur in the knee, elbow, and tibio-tarsal joint.

Deformities also occur at the joints, due usually to osteophytic growths at the epiphysis. Such growths may constitute obstacles to motion, and may even give rise to ankylosis or to secondary changes within the joints, as well as to muscular atrophies or even an arrest of development in the affected limb.

Kidneys.—The existence of disease in the kidneys of syphilitic infants is fully established by accurate observation, though our knowledge on the subject is still small. These same affections show themselves at a more advanced age, but our knowledge of their history in the later cases is no more complete than in infants. Present knowledge on the subject is stated by Fournier[1] in the following propositions, here somewhat abbreviated:

1. Different forms of nephritis have been observed.
2. A common characteristic unites all these forms: it is the chronic character of the renal changes. All consist of torpid and slow degenerations, where the inflammatory element finds no place.
3. Two forms appear to be more common than the others,—*parenchymatous nephritis* and *amyloid degeneration.*
4. The interstitial form, with small contracted kidney, seems more rare.
5. The gummatous form, at least that with isolated and circumscribed gummy nodules, may be considered exceptional.

M. Ernest Dupré reports[2] a suggestive case observed in the hospital Trousseau, of a girl of fourteen with interstitial keratitis and periostitis of the lower end of the humerus, which recovered under iodide of potassium and mercurial inunctions. Her father was an old soldier of Africa, and of nine children only two had survived infancy. This girl was albuminuric at the time of her admission to the hospital. During the first week of her stay in the hospital, while she was not under antisyphilitic treatment, the slight albuminuria had increased in a notable proportion. When the iodo-mercurial treatment was instituted, the albuminuria diminished rapidly and in five days disappeared.

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[1] Fournier, p. 357.

[2] La France Médicale, March 13, 1888.

Diagnosis.—It too often happens that information cannot be obtained concerning the condition of the parents before the birth of the child. If obtainable, such information is, of course, conclusive. In its absence information in regard to the family may furnish most valuable diagnostic points. A suspicious fact in the history of father or mother may sometimes be elicited, as the loss of the palate or some obscure nervous disorder. A large infant mortality is very suspicious. Of course the causes of death among infants are too many to allow that fact alone to carry very great weight, but sometimes the mere statement of the deaths is almost enough to make a certainty of what would otherwise be mere suspicion. For instance, a young girl presented with suspicious lesions. She was the youngest of fifteen children: of the first eight, five had grown up well and strong and three had died in childhood of ordinary children's diseases. Then followed a series of six still-births and deaths in early infancy, and, last of all, the patient, with symptoms and personal history pointing strongly to inherited syphilis.

A careful examination of the brothers and sisters will often throw light upon the case. In an heredito-syphilitic family the symptoms are seldom identical in different children. If a case presented for examination is simply suspicious, the brothers and sisters may possibly exhibit signs that are more definite. The Hutchinsonian teeth are often confined to a single member of the family, and that generally the eldest.

Examination of the cornea may not always show signs of a previous keratitis, but in a large number of cases a history will be given to the effect that at such a date the patient suffered with his eyes and was actually blind for a series of weeks or months.

The eruptions of the early stages of the disease do not generally leave cicatrices, or at least do not leave characteristic cicatrices. The ulcerations about the mouth, however, which are so frequent that few syphilitic infants escape them, often leave behind them delicate scars, which are of a certain value as diagnostic marks during the later years of childhood. These scars are most common and most characteristic at the commissures of the lips, when they are often so delicate as to be visible only on close inspection. They are quite characteristic of syphilis, but may, of course, owe their origin to any ulceration at that spot.

Cases of periostitis in children are suspicious, especially if the periosteal inflammation is situated at the lower end of the humerus or on the anterior border of the tibia. Cases in which there is simultaneous periostitis of several bones are particularly suspicious. If the two tibiæ and one or both humeri are diseased at the same time, syphilis may be assumed as practically certain.

Prognosis.—The prognosis of late congenital syphilis is in great measure the same as that of tertiary syphilis. There is a special tendency to return, and we can never pronounce a patient definitely cured, although many individuals go for long periods without a return of their complaint in any form, and there is reason to believe that certain heredito-syphilitics who manifest late symptoms are definitely cured.

As to the separate attacks the prognosis is very different. The prospect of recovery from any individual attack is always good. Of course actual loss of substance cannot be made good, whether that occurs in integument, bone, or mucous membrane. But if the patient will submit to proper treatment, the progress of the disease can usually be arrested and the loss be confined to the portion actually destroyed at the time when first seen.

Treatment.—I am inclined to think that the necessity for treatment in these late cases is not fully appreciated. They need to be treated in the same way and as carefully as the acquired cases, and perhaps even more persistently. Treatment is not universally successful, but in many cases its results are brilliant, and I think I may say the success depends in great measure upon the faithfulness with which it is carried out. Practically the same plan that is pursued in so-called tertiary disease in the adult is adapted to these late inherited lesions,—that is, a treatment into which the iodides enter largely in conjunction with mercurials. The iodides are especially useful, either alone or in combination with mercury, and in some cases they need to be given in large doses.

Too often these cases are regarded as scrofulous, and are dosed *ad nauseam* with cod-liver oil, a remedy which is of great value as an adjuvant to meet certain exigencies, but which falls far short of the brilliant results sometimes achieved by the antisyphilitics. Sea-air is also prescribed, but its value is not great. Tonics of all sorts are useful to a limited degree.

There is every reason to believe that very great influence may be exercised upon the growth of heredito-syphilitics by appropriate treatment. Fournier speaks emphatically upon that subject in mentioning the change wrought in a young girl, and its influence has been nearly as marked in cases of my own. Bone actually dead cannot be revivified,—it must be mechanically removed,—but the morbid process can be controlled and further destruction prevented. There is reason to believe that energetic treatment may, at least in some cases, prevent such misfortunes as the loss of hearing, if it is undertaken sufficiently early.

RACHITIS.

By THOMAS BARLOW, M.D., F.R.C.P.,

AND

JUDSON S. BURY, M.D., M.R.C.P.

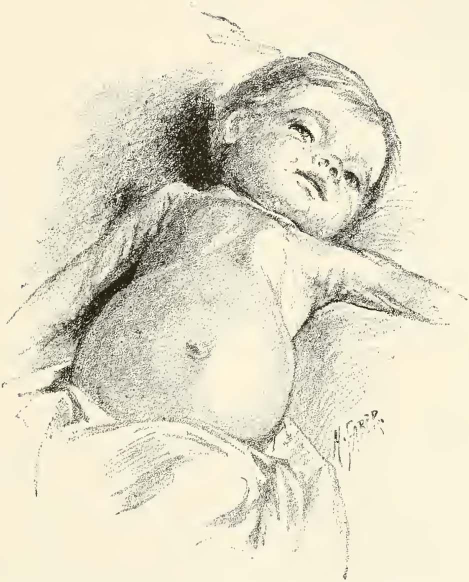
THIS disease, which is probably coeval with civilization, was first accurately described by Dr. Glisson, in the middle of the seventeenth century. The term rachitis was given by Glisson as the classical equivalent of the common West-country name of the disease rickets, and he also desired in this term to emphasize one of the special manifestations of the disease,—viz., deformity of the spine (*ῥάχις*, the spine).

Definition.—Rickets is a disease of infancy, in which there is a varying amount of general impairment of nutrition, but which is mainly characterized by definite alterations in the growing skeleton, the essential part of which consists of overgrowth and imperfect organization of some of the developing elements, resulting in altered consistence, temporary or permanent arrest of growth, and some deformity which tends towards spontaneous involution.

Symptomatology.—The earliest manifestation of rickets consists in beads at the junction of the ribs with the costal cartilages. Collectively these constitute the so-called rickety rosary. The beads are generally less marked in the upper one or two than in the lower ribs, being most obvious about the fifth and sixth. Such beads may not be very apparent unless the child is thin, but they can be easily detected by touch.

With regard to the period when these beads first appear, they may very often be detected at three months, though they are seldom massive at that age; but they may be identified as early as one month, and we have found them on dissection of still-born children.

In some cases simultaneously with the beads, in others before and in others after the appearance of the beads, there may be found certain early skull-changes, which may now be considered. First the free margins of the flat bones of the skull are unduly soft, and in cases which have been traced onwards for several months from birth these parts of the bones are found to be late in ossifying, and small islands of bone remain for a considerable time in the membrane between the parietals and the occiput.



CHILD, THE SUBJECT OF RICKETS.—Head shows bossy frontals. Thorax shows anterior convexity (sternum and costal cartilages), lateral grooves, and transverse constriction. Abdomen large.

But, besides these areas of delayed ossification, other changes may sometimes be found; the occiput and the parietals may yield to the pressure of the finger like parchment, and round spots of local thinning may be detected on these bones and even exceptionally on the frontal. To these spots, as well as to the general abnormal flexibility and parchment-like character above described, Elsässer, who first discovered the condition, gave the name of *craniotabes*.

Such spots are most frequently met with about the third month; they may be found up to the eleventh, but do not appear after that period.

Slight beads on the ribs, with or without craniotabes, lasting for a few months and then subsiding, may be the only symptom of rickets demonstrable on external examination. In like manner craniotabes—in so far as it means a flexible occiput or parietal—may exist for a time and then pass away without even beads appearing on the ribs or any further sign. There is, in fact, scarcely any chronic disease which shows more quantitative variations than rickets, going down to a vanishing-point which needs the greatest care in order to establish its existence, and having in its mildest form a duration perhaps of only a few weeks. But, assuming that the case increases in severity so as to become recognizable even to the merest tyro in medicine, let us now consider the different changes in the skeleton so far as they are identifiable clinically.

Skull.—Whilst the margins of the bones still remain semi-membranous, and the craniotabetic spots are still to be felt at the postero-lateral parts of the skull, hyperplastic changes become manifest in the forepart. Symmetrical low lenticular swellings form on the frontal and parietal bones in front of and behind the anterior fontanel respectively. These masses of soft, vascular, bony growth shine through the thin pale scalp of the infant, and in some cases cause a characteristic pale-bluish-looking swelling. There is no local heat or tenderness over these areas, but there is probably some discomfort in the early stages, as infants so affected, besides showing much irritability, sometimes throw the head about a great deal and bore the pillow. In aggravated cases these bossy swellings increase in size, but maintain more or less symmetry, and other swellings arise below and around the parietal eminences, on the upper part of the occiput, and on the temporal regions. Band-like elevations may also be traced near the sutures. All these new superposed osseous growths, if not absorbed, gradually become more or less diffused and organized, and thus give rise to the various characteristic forms of the rickety skull. Of these there are two principal shapes to be noticed: the commonest shape presents a broad square forehead, with the frontal eminences or the bone around strongly developed; the crown is flattened, as though it had been subjected to pressure from above, but still shows some indications of the original four bosses, with a broad median groove and a ridge on each side of it. The parietal eminences and the occipital protuberance are also well marked. The above type of skull, when carefully examined, is found to be totally different from that of a hydrocephalic skull: an antero-posterior tracing of the outline of this form of skull is polygonal, whilst that of a hydrocephalic skull is almost circular. The second type of rickety skull is one elongated fore and aft, or markedly dolichocephalic; the frontal region, not very broad, is prominent in the middle portion, the top of the skull is also flattened, and the upper part of the occiput projects considerably. Other rickety heads show marked asymmetry, especially in the posterior part, and this is occasionally accompanied by compensation in the forepart, the frontal region being prominent on the same side as the flattened parieto-occipital region, and *vice versa.* Other cases show the whole occipital region flattened so as to appear nearly vertical on side view.

Whatever the shape, a rickety head is generally larger than the head of a healthy child of the same age. It may indeed be found that the circumference of the head of a rickety child is greater than that of its thorax.

All forms of rickety skull agree in late closure of the anterior fontanel. Instead of being obliterated by eighteen months, it may remain in extreme cases still obvious till the child is five years old.

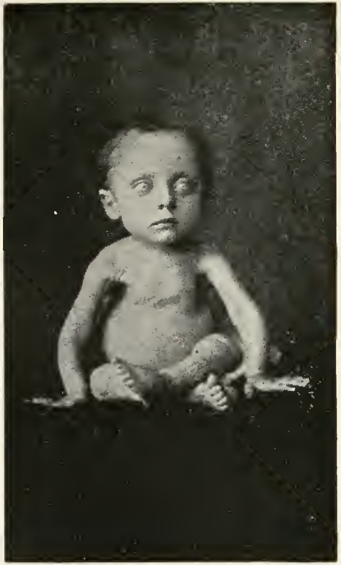
The sutures (except the medio-frontal, which may be prematurely bridged over with osseous material) are generally delayed in their union, and the bony edges are thick and irregular. Over the scalp large, prominent veins are frequently to be seen; these are often partly contained in deep grooves, which may be mistaken for open sutures, especially in the temporal region: the grooves arise in connection with the formation of the deposits of vascular bone just described.

With respect to the face generally, it often looks small in contrast to the massive frontal region. The bones of the face most obviously affected by the rickety change are the maxillæ. As Fleischmann has pointed out, the alveolar border of the upper jaw tends to assume a beak-like shape, the antero-posterior axis being lengthened, whilst the outline of the lower jaw becomes somewhat polygonal and its anterior surface turned slightly inward. Modifications of dentition vary according to the period at which the active phase of rickets becomes manifest. If it be early, there are three ways in which the modifications may occur. (1) The teeth are late in their eruption: it is by no means uncommon to find a rickety child twelve months old with only a single tooth. (2) The teeth are cut "cross,"—that is, they appear in wrong order. (3) They soon become carious, and are often shed early.

Chest.—We pass now to the changes in conformation of the chest. The rickety rosary has been already referred to. The enlargements which constitute the rickety beads vary in size from a thickening which can scarcely be felt externally, to a mass the size of a cherry.

The anterior beads are generally symmetrical on the two sides. As before stated, they may be identifiable even at birth, they become distinct at three months, and thenceforward increase in a typical case of average severity up to the end of the second year, after which they generally recede. They are rarely found in children over five years old, except in severe recrudescences of the disease. No vestige of them remains in adult life.

Photo. No. 1.



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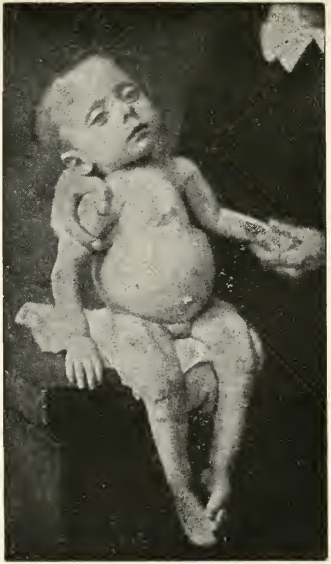


Child, aged Five Years, the Subject of Rickets.—Photo. No. 1 shows ordinary sitting attitude of advanced rickets,—lower limbs crossed, head and trunk partly supported by the hands. Deformity of upper limbs corresponding with mechanical pressure. Photo. No. 2 shows slight anterior convexity of femora. Both photographs show anterior convexity of thorax, with transverse constriction, also the large abdomen.

Besides these anterior swellings, there are also to be found, in severe cases, backward projections from the ribs, close to the angles; they differ from the anterior beads in the following respects: they are less symmetrical; they vary in position on successive ribs; sometimes they are present on only one side, or if on both sides they occur at varying distances from the middle line; finally, the projections are angular rather than nodular.

The changes in the contour of the thorax, as a whole, are the following: First, a slight groove is to be felt, and often to be seen, immediately anterior to the row of beads: more obvious is a broad, shallow depression, beginning outside the nipple on each side, extending obliquely from above downward and outward, and situated immediately behind the beads. This growth is deepest in the upper and anterior part of the axillæ, but below stops short of the false ribs. Second, there is a transverse groove (Harrison's sulcus) extending from the line of junction of the body of the sternum with the ensiform on either side outward to the posterior axillary border. In severe cases this groove is deepened during each inspiration. Third, there is increased convexity of the costal cartilages, which with the sternum are carried forward and present a broad rounded front. The horizontal outline of a typical rickety thorax, as taken by a crytometer-tracing through the sterno-xiphoid articulation, presents a figure not unlike that of the periphery of a violin; the broadest portion being posterior, the narrow, rounded portion being anterior, and the constriction corresponding with the lateral grooves.

The pigeon-breasted chest is often attributed to rickets, but it is doubtful whether this is necessary for its production, although the two may concur; the essential features of this form of thorax are that the outline of the horizontal section approximates to the triangle, that the ribs from the angles forward lose their normal convexity and become straightened, and that the sternum is carried forward. It is significant that this form of chest may often be found *without any beads;* that it is very rare in children under twelve months old, except in those who are the subjects of atelectasis and congenital malformation of the heart; and that it is most commonly found in children over two years of age who have suffered from prolonged whooping-cough or some other chronic respiratory trouble interfering with the entrance of air and leading to collapse.

Back.—The back, in the earliest phase of the disease, seems to yield when the child is placed in the sitting posture. The dorsal spines project backward, not abruptly as in kyphosis from caries of the spine, but form a gradual rounded convexity. If the child be laid on its belly and gentle traction be made on the legs, it is easy to see that in the early stages of the disease the excurvation is not a persistent one. The same remark may be made about the early lateral curvature which is often present.

But even before the end of the first year of life, if a child suffering from moderately severe rickets is often kept in the sitting posture, a permanent deformity may ensue, varying from a gradual curve up to a rounded gibbosity and accompanied by some degree of lateral modification. Apart from the cases resulting from pleurisy, and from infantile paralysis affecting the muscles of the back, the commonest cause of lateral curvature in young children is rickets; but at the age of puberty, and even in children of six or seven years of age, this condition seems to arise independently of the diathesis in question.

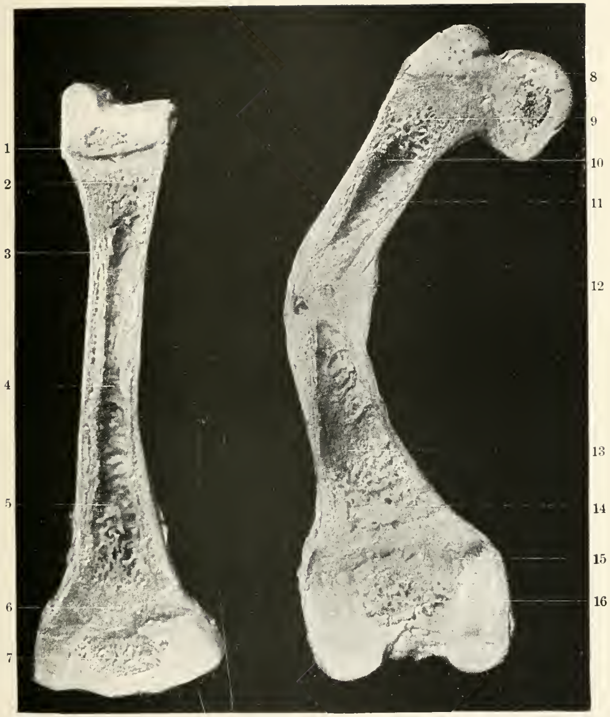
Shoulder-Girdle.—The changes in the *shoulder-girdle* are marked only in the severe cases. The scapula may be found altered in such a way that the anterior surface is more concave than normal, the supraspinous fossa being somewhat depressed and the inferior angle curved forward. Thus the infraspinous fossa, instead of presenting the normal flatness, is found somewhat convex backward. The lower angle and the axillary border become slightly thickened.

The clavicle is sometimes thickened at its sternal end, and at a varying distance—often near the outer extremity of the attachment of the sterno-mastoid—there is situated a swelling due to green-stick fracture and callus. This swelling is often symmetrical on the two clavicles.

The whole of the pelvic changes are difficult to appreciate during life, though it is easy to make out the thickening of the crista ilii and in some cases the narrowing of the pubic arch. In a very extreme case of late rickets under the care of one of the writers, there was a considerable amount of lumbar lordosis and a certain amount of what may be called "crumpling" of the ossa innominata; in other words, the upper portions of these bones were bent inward, with some thickening of the crista ilii, the ischial tuberosities were flattened, broadened, and somewhat turned inward, and the intermediate portions of the ilia were unduly convex backward, whilst the coccyx was quite horizontal. The change in shape of the ilium was quite comparable with that of the scapula above described.

Limbs.—The earliest naked-eye changes in the *upper limbs* are observed at the wrists, the lower ends of the radius and ulna being larger than normal. The enlargement is no doubt primarily at the junction-area of the shaft with the epiphysis, but the epiphysis itself becomes too big. This change is seldom obvious to naked-eye inspection earlier than the third month. It is often considerable by the tenth month, but in cases of average severity rarely increases after the end of the second year. It may, however, be still active in very aggravated cases at the age of four or five years, and some vestiges may remain as late as puberty.

To a much less extent enlargements may be found at the lower end of the humerus, and to a still less extent at the upper end of the humerus and the upper end of the radius and ulna. Changes in the shape of the shafts are not met with in the early stage, but they are found in the aggravated cases at a later period, when they seem to bear some relation to the amount of pressure to which the bones are subjected either in crawling or in the half-squatting attitude often assumed by the rickety child. In this attitude the child sits with its lower limbs crossed, leaning forward, part of the



Tibia with the Principal Lesions of Rickets.

Femur showing all the Principal Lesions of Rickets.

1, zone of proliferating cartilage; 2, ossifying zone (spongioid bone); 3, cortical layers,—outer layers (recent) of soft, limeless bone, inner layers (older) of calcified bone; 4, rarefaction of medullary portion; 5, ossifying zone (spongioid bone); 6, zone of proliferating cartilage; 7, diffuse ossifying centre of epiphysis; 8, zone of proliferating cartilage; 9, ossifying zone (loose spongioid bone); 10, rarefaction of medullary portion; 11, cortical layers,—outer layers (recent) of soft, limeless bone, inner layers (older) of calcified bone; 12, green-stick fracture, buttresses of soft, limeless bone on anterior surface and on posterior surface; 13, rarefaction of medullary portion; 14, ossifying zone (spongioid bone); 15, zone of proliferating cartilage; 16, diffuse ossifying centre of epiphysis.

weight of the head and trunk being supported by the outstretched palms, which are placed flat on the bed. This attitude explains the undue concavity sometimes present on the whole inner aspect of the upper limb. But the change in shape is not generally expressed by a single curve in one direction: even in the living body it is easy to detect, in both the arm and the forearm, that the bones have undergone more or less of a spiral distortion, the maximum changes being near the extremities.

In rare cases very early complete fractures occur, but the writers hold that these are present only in infants whose rickets has started in early intra-uterine life. More common than the complete fractures, and occurring later, are the so-called "green-stick" fractures, in which an abnormal projection, sometimes uneven, sometimes rounded, but rarely splintered, is found on some part of the shaft, without any loss of continuity. The violence giving rise to the green-stick fracture may in aggravated cases be so slight in amount as entirely to escape notice.

Occasionally slight thickenings may be detected near the growing ends of the metacarpal bones and the phalanges.

The changes in the *lower limbs* have much in common with those of the upper, but they are not obvious to the naked-eye inspection quite so soon. The lower end of the tibia may be considered the place of election. In severe cases it never escapes, and in slight cases it may be the only bone of the lower extremity which shows enlargement. As in the lower end of the radius, the rickety change consists in thickening at the junction-area and enlargement of the whole epiphysis. The upper end of the tibia is the one which comes next in order as to frequency of enlargement, and with it the lower end of the femur, but much less often the upper end of the femur and also the two extremities of the fibula. The changes in the shafts are no doubt more marked in children who have got about either by crawling or walking, but very decided changes may be found in infants who have never borne any weight on the lower limbs. The simplest and most common deformity is a slight concavity of the tibia on its inner surface,—so that the two tibiæ approximate at the knees and also at the ankles, but are separated from each other between the extremities. If the child is kept horizontal, no further alteration in the axis of the limbs takes place. But if the rickety infant is carried in the arms to any extent, and if the disease is actively progressive, the femora become arched forward and a marked convexity forward is also manifest in the lower third of the tibiæ; in some cases, when the child is allowed to be on its feet, an outward curve is added to the forward one in the upper third of the thigh, and the forward and outward deformity becomes still more exaggerated in the leg. Bowing often occurs to an unequal extent in the two legs; less common is knock-knee, and occasionally there is knock-knee on one side and bowing on the other; still less common is the condition in which the tibia yields in a backward direction along the line of junction of the upper epiphysis.

Ligaments.—The *ligaments* in the neighborhood of many of the joints also suffer; owing to the active changes in the ends of the bones to which they are attached, their nutrition is damaged, and they subsequently yield, and thereby contribute to the looseness of certain joints. The commonest deformity of the foot is talipes valgus, from yielding of the ligamentous structures of the foot, and this as well as knock-knee can be temporarily overcome by simple traction.

Muscles.—The *muscles* also in many situations are demonstrably thinned and poorly nourished. Here may be considered a symptom which varies within wide limits in different cases,—viz., *tenderness* of the limbs. In the slight cases, and even when the disease is well marked, with considerable deformity, this symptom may be wanting, or present for only a short time during the active phase. The presence of tenderness probably explains in part the dislike for movement exhibited by rickety children.

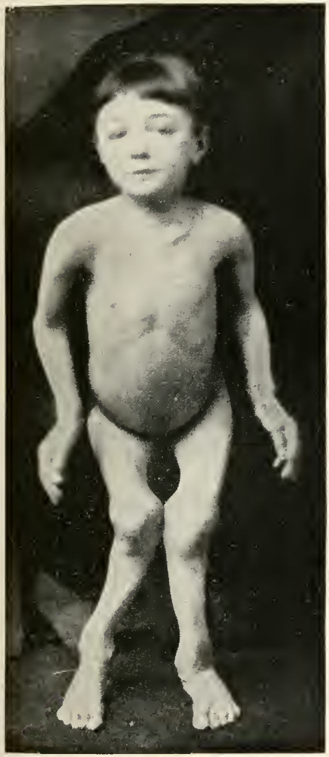
It seems to be in the main a bone-tenderness, and to reach its maximum in the epiphysial-junction region, but it is often very ill defined. When it occurs with very great severity and is accompanied by powerlessness of limbs, the writers believe it is often due to the supervention of sub-periosteal blood extravasations (see article on Scurvy). Along with the tenderness of limbs ought to be mentioned the irritability which sometimes accompanies the acute phase of the formation of cranial bosses, and which is possibly due to the overgrowth of vascular osteoid material and the accompanying stretching of the pericranium.

Skin.—The skin in slight examples is very little altered, but in the active phase there is generally excessive sweating, most marked on the head. This is, indeed, one of the earliest accompaniments of rickets, and may be noteworthy before the beading becomes prominent. It is possible that the head-sweating bears some relation to the hyperplastic changes taking place in the cranial bones at an early stage of the disease.

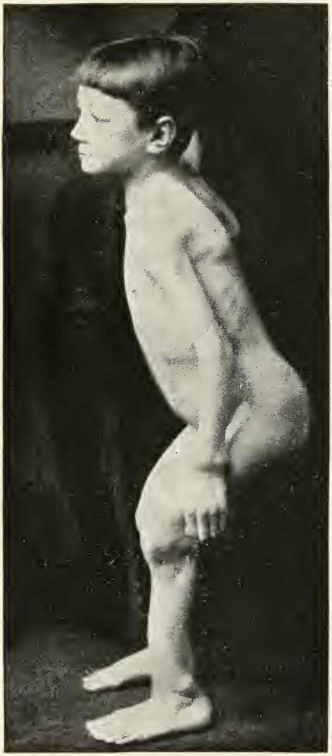
With respect to *pyrexia*, it may be stated (1) that in many cases it appears to be completely absent, (2) that in other cases it is certainly absent over considerable periods whilst rickets is still progressive, (3) that intercurrent catarrhs may readily give rise to pyrexia, and (4) that during the early active phase of a severe case it is possible that some pyrexia may occur in direct relation to the bone-change. (For the occasional pyrexia of so-called acute rickets, see article on Scurvy.)

Ordinary cases of rickets show little or no pallor; in long-standing and severe cases there is not only anæmia, but also considerable pigmentation, especially on the extensor surfaces. In regard to the subcutaneous fat, it is well known that "fat rickets" is commoner than "thin rickets." A great many slightly or moderately rickety children are, indeed, stouter than healthy children of the same age. In some prolonged and severe cases, no doubt, emaciation supervenes, but the writers are of opinion that rickets is rarely initiated and rarely very active during a period when severe wasting of the tissues of the body from any cause occurs.

Mucous Membranes.—In a typical rickety chest there is no doubt a



Case of Late Rickets (aged Eleven Years). —Humeri thickened and rounded, especially near insertion of deltoids; lower epiphyses of radii and ulnæ much enlarged. Femora show the neck nearly horizontal, the shafts convex forward, the lower epiphyses enlarged. Tibiæ show large lower epiphyses, the shafts convex forward in lower third, slightly concave forward in upper third.



Case of Late Rickets (aged Eleven Years). —Shows enlargement of lower epiphyses of radii and ulnæ, thickening of crista ilii, and bulging backward of ossa innominata, lumbar lordosis, enlargement of lower epiphyses of tibia and fibula, slight anterior concavity of tibia in upper third and convexity in lower third.

tract of collapse corresponding with the grooves and some emphysema corresponding with the anterior convexity (see section on Morbid Anatomy). Moreover, there is proneness to bronchial catarrh, and this is specially liable to give rise to collapse and broncho-pneumonia; but we are not aware that there is anything specially belonging to rickets in these affections.

The alimentary tract is in like manner easily prone to catarrh. Some gastro-intestinal disturbance precedes the obvious manifestations of rickets in a large number of cases, but this is often subacute in character. The commonest manifestation is the occurrence of foul-smelling fæces, white, brown, or green in color, with or without frothy mucus. The number of daily evacuations is not necessarily increased, and even alternating constipation may be observed, or the passage of hard, compact masses of undigested casein. The abdomen is often big in the active stage of rickets. Several factors contribute to this, of which the most important are the existence of flatulent distention of the bowels, the flabby muscular parietes, and the contracted chest, with the consequent lowering of the abdominal viscera.

Liver.—Besides being depressed, the liver is sometimes distinctly enlarged, with a smooth surface and a rounded edge. More important is the enlargement of the spleen which is sometimes present to a moderate degree. In rare cases it attains a great size, extends as low as the iliac crest, and crosses the middle line.

It is to be noted (1) that the severe cases are almost invariably associated with anæmia, which is sometimes profound, and of the chlorotic variety. The blood shows diminution of red corpuscles, but no marked increase of leucocytes. (2) The amount of *rickety bone-change may be very slight indeed.* (3) In the great majority of cases of rickets, even when very severe, splenic enlargement is not present. It appears to us, therefore, that the enlargement of the spleen and the accompanying anæmia are not essential to rickets, but that they belong to a separate cachexia.

Urine.—A complete and exhaustive examination of the urine in rickets during the different phases of the disease is still a desideratum. The early analyses of Marchand, according to which six times the normal quantity of lime salts was found, have not been verified by later observers. Indeed, the differences in respect to earthy phosphates between the urine of healthy and that of rickety infants appear to be quite unimportant. No modern observer has been able to establish the existence of lactic acid in the urine of a rickety subject.

NERVOUS DISTURBANCES.—Sooner or later marked nervous disturbances occur in most cases of severe and in many cases of moderate rickets. Perhaps the earliest and one of the most constant of these is the undue irritability in consequence of which rickety children persistently throw off the bedclothes during sleep.

Laryngismus stridulus, though it is not absolutely restricted to rickety children, and is not present in every case of rickets, is a very common complication. It may be defined as a sudden arrest of respiration, followed by a long-drawn crowing sound due to inspiration through a narrowed glottis. The attacks are frequently only of a few seconds' duration, but during the period of arrested respiration there is fixation of the diaphragm and of the respiratory muscles, and it is by no means uncommon for the thumbs and fingers to become tightly flexed on the palm, for a slight degree of cyanosis to occur, and even for a convulsion to follow rapidly. Attacks of laryngismus often occur on waking from sleep, on sudden movement or fright, as when "a child is put out," sometimes in connection with swallowing efforts, and after sudden exposure to cold gusts of air.

Although in the great majority of cases harmless, occasionally laryngismus is suddenly fatal. Laryngismus stridulus was believed by Elsässer to depend on craniotabes, and the attacks he considered were brought about by repeated localized pressure on the brain-substance through the softened areas at the back of the skull. But it is probable that the only link is that they are each manifestations of rickets, for, apart from the fact that the two conditions often occur independently, laryngismus is more common after nine months than before, whilst craniotabes is pre-eminently an early manifestation.

Convulsions.—It was pointed out by Jenner, and has been abundantly demonstrated by Gee, that the eclampsia of infancy has often a close relation to rickets. It is not the fits of the first three months of life, but those of the latter part of the first year and of the second year, which are so frequently associated with rickets. Such fits, no doubt, arise at times in connection with the irritation of teething, and it is in rickety children *par excellence* that teething gives rise to irritation. But other kinds of peripheral irritation also seem to determine the occurrence of fits, especially the gastro-intestinal troubles which are so common in rickets.

Tetany.—The condition now commonly designated as tetany occurs in adult life after some of the fevers and after parturition, and it also occurs in childhood apart from rickets, but when present in infancy it is almost invariably in rickety subjects.

As the manifestations of *tetany* in infancy and its relations to rickets are often ignored, it seems to deserve a more detailed description than was necessary with the preceding nervous affections. Tetany may be defined as a more or less continuous tonic spasm of certain groups of the limb-muscles, lasting for varying periods and generally symmetrical. A typical case is the following: a rickety child aged twelve months, having had recently several attacks of laryngismus and passed some more than usually fetid stools, wakes up in the morning with painful contractions of both hands.

The most characteristic contraction is that described by Trousseau as simulating the accoucheur's hand. There is slight flexion of the wrist and spasm of the interossei. The thumb is applied with its tip against the middle phalanx of the ring-finger, whilst the fingers present flexion of the metacarpo-phalangeal and extension of the interphalangeal joints, and some of the fingers overlap one another. In a severe case there may be redness and slight œdema of the back of the wrist.

Both hands are affected, but not with equal severity. These contractions, especially at the outset, are evidently painful. They may continue unchanged for some hours and even during sleep, and with some remissions they may be present for several days and even weeks. During the remissions it was pointed out by Trousseau that pressure on the main vessels or nerves of the limb may re-excite the tonic contraction. But this is not always the case, and, moreover, during the early and active phase of the affection a great variety of stimuli seem to reinduce the spasm. The association with laryngismus is very remarkable; indeed, not only at the onset, but frequently also during the course of the affection, a fit of laryngismus marks an exacerbation of the tetany.

The lower limbs are often affected along with the upper, but to a much less extent. There may be tonic extension and inversion of the ankle, but the characteristic change is *extreme flexion of the toes*, which sometimes overlap one another. The forepart of the sole is often contracted so as to become remarkably concave, with a median furrow, due to the attempted approximation of the outer and inner margins of the foot. The dorsum, like the wrist, may be a little red and shiny, perhaps in consequence of the mechanical interference by the severe tonic spasm with the circulation in the part. Very rarely indeed a little opisthotonus occurs. During the continuance of the spasm of the extremities, and for a time after its subsidence, there is a remarkable modification of the neuro-muscular irritability. Several years ago one of the writers discovered this in regard to the facial muscles. This can be easily demonstrated in a child suffering from tetany by drawing the forefinger sharply over the skin in front of the ear, when an instantaneous contraction (very like that produced by the constant current) will be found to occur in the muscles supplied by the facial nerve. In the early active phase of tetany this is very striking indeed, but as the affection is gradually subsiding the irritability becomes less pronounced. The facial muscle which is the last to show it (in other words, which is most irritable) is the orbicularis palpebrarum.[1]

Other manifestations of neuro-muscular irritability have been pointed out by several observers, and Erb has shown that not only is there increased electric excitability of nerves to both faradism and voltaism, but also that the mode of reaction to the voltaic current is altered, the earliest contraction occurring to positive instead of to negative closure. Further, with both positive and negative opening and closing, there occurs a prolonged contraction or "tetanus" which is very remarkable.

Morbid Anatomy.—Under the morbid anatomy are to be considered (1) the naked-eye and (2) the histological changes. In regard to the former it should be noted that not only are there variations according to the age of the morbid process, but also that the different elements of the rickety change show great difference in their grouping.

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[1] See Abercrombie on Tetany in Young Children, 1880.

Let us consider first the *long bones of the limbs.* The simplest, earliest, and frequently the only detectable change is the enlargement of the junction-area between the shaft and epiphysis; this causes a slight but perceptible bulging on the outside, and, when a vertical section is made, it is seen to be composed of a cushion of cartilaginous material of a somewhat bluish color, and more gelatinous in consistence than the epiphysial cartilage; this cushion passes insensibly into the epiphysial cartilage on the one side, but towards the shaft presents an irregular and digitate border. This may be the only abnormal condition present in a given bone, and, although symmetrical on the two sides of the body, if there be two epiphyses to the bone the change may be more obvious at one junction-area than at the other, or may be present at one junction and not exist at the other.

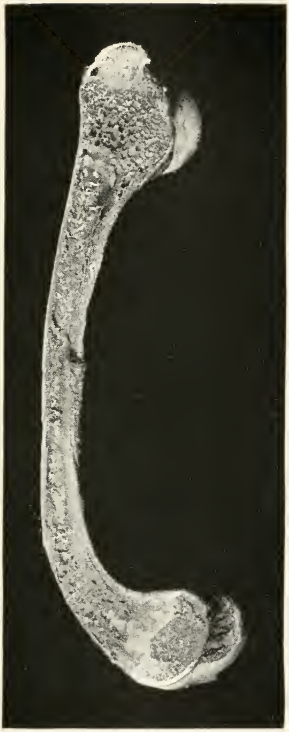
Passing from this simple, constant, and fundamental change, we may meet with changes varying greatly in degree and extent. The further changes may be conveniently considered in regard to the different parts which make up the whole bone.

First, with regard to the *junction-area* itself, there are great variations not only in respect to the width of the cushion, but also in the degree and in the irregularity of the subsequent calcification.

The width may vary from a line or two to half an inch. As to the calcification, tracts of granular earthy material are seen invading to a varying degree the proliferated cartilage. There is, indeed, as Sir William Jenner has well said, an extensive preparation for the processes of ossification, with an imperfect performance thereof.

Secondly. The ossifying centre of the epiphyses may present a diffuse form, being much larger than natural, more vascular, and more spongy in character. One result of this condition, in some cases, is a general enlargement of the whole epiphysis, as distinguished from other cases in which the enlargement is limited to the junction-area. Also it is to be noted that there are great differences in the rapidity with which the union between shaft and epiphysis takes place; in some, this occurs at a much earlier period than in the healthy bone, hence growth in length is arrested, and it is in these cases that dwarfing takes place. In other cases the union is not premature, and, although the rickety changes may be considerable, complete involution takes place and the bone attains its natural length. It is often found, in the course of osteotomies performed in cases in which the rickety process has come to an end, but which present permanent deformity, that considerable sclerosis exists in this region.

Thirdly. Turning to the shaft of a long bone, we may first note that the periosteum strips off more easily and shows larger vessels than in a natural bone. Great variations as to the character and amount of the subperiosteal bone occur: in some cases there is no excess, in others there are several layers. Virchow has counted six to ten, but usually there are only



Femur from Case of Late Rickets (aged Eleven Years).—Shows at the upper end the neck nearly horizontal, loose, spongioid ossification; on the concave posterior surface of the shaft several layers of new bone, forming buttress, some rarefaction of the medullary portion of the lower third of the shaft, with thinning of the anterior cortex; at the lower end large islands of proliferated cartilage.

two or three. They are often of different structure: the outermost layer may consist of a semi-elastic callus-like material entirely devoid of lime salts, not unlike in character decalcified bone: this is the so-called "spongioid bone;" when it exists in great excess it gives rise to the *limeless-bone* form of rickets. The deeper layers of the subperiosteal bone are more fibrous in character, while the deepest are more completely impregnated with lime salts than the superficial ones.

Fourthly. Generally in rickets the medullary portion of the shaft is more vascular than natural, and presents an appearance approximating to that of red-currant jelly; on washing away the marrow, the trabecular framework is seen to be rarefied, the interspaces being much larger than normal. The rarefaction may be so extreme that it becomes the predominant feature.

The variations above described will account in some degree for the marked differences observed in the tendency that rickety bones show either to yield or to break. First, with respect to *yielding*, the deformities briefly indicated in the section on the symptomatology are chiefly exaggerations of the normal curves. Such exaggerations have been attributed in some degree to muscular traction, but the writers are of opinion that Sir William Jenner's view is the correct one,—viz., that they may be mainly referred to the influence of pressure or weight acting on the growing weakened bone.

Fractures are commonly met with in examining rickety bones even when not suspected during life. The common form is a limited green-stick variety which usually occurs at the point of the greatest curvature of the shaft. On making a section of the shaft, the girth may or may not be increased, though a certain amount of buttressing is usually found either crossing the medullary cavity, or, when the fracture is impacted, surrounding the broken ends and filling up the concavities. Another quite distinct form of fracture is rarely met with in very young infants, and this is described in a subsequent section.

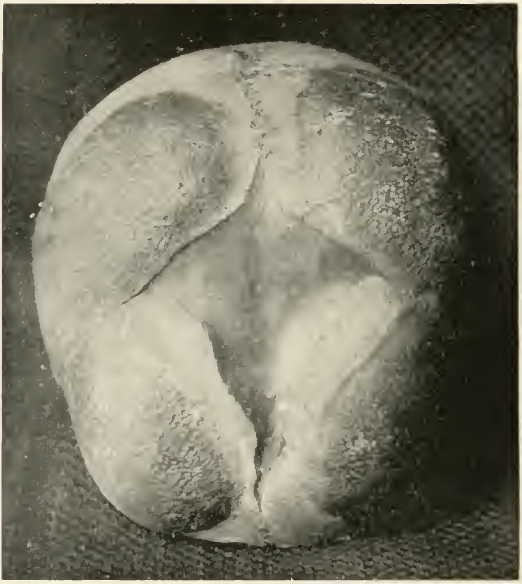
Thorax.—To the points already alluded to in the symptomatology it may now be added that the beads are much more marked on the pleural than on the external surface, and this applies to the lower as well as to the upper ribs. The minute structure of the beads will be discussed in the histological section. The posterior beads before described are now found on section to be due to partial fractures with more or less callus; sometimes there is no change on the pleural side of the rib to be made out, while in other cases a slight groove indicates the place of the posterior projections.

As to the grooves, it will be found on dissection that the transverse groove corresponds not to the attachment of the diaphragm, but to the upper limits of the stomach, liver, and spleen, and that it is often not quite symmetrical on the two sides. The chief factor in the production of the grooves seems to the writers, following the teaching of Jenner, to be atmospheric pressure: this acts with greater effect on the rib than on the cartilage, the former in rickets being softer than the latter, especially in the part just posterior to the beads, where the lateral groove is formed. The eversion of the lower parts of the thorax below the transverse grooves corresponds with the position of the unyielding abdominal viscera, and the slightly greater prominence of the left front as compared with the right is due to the underlying heart.

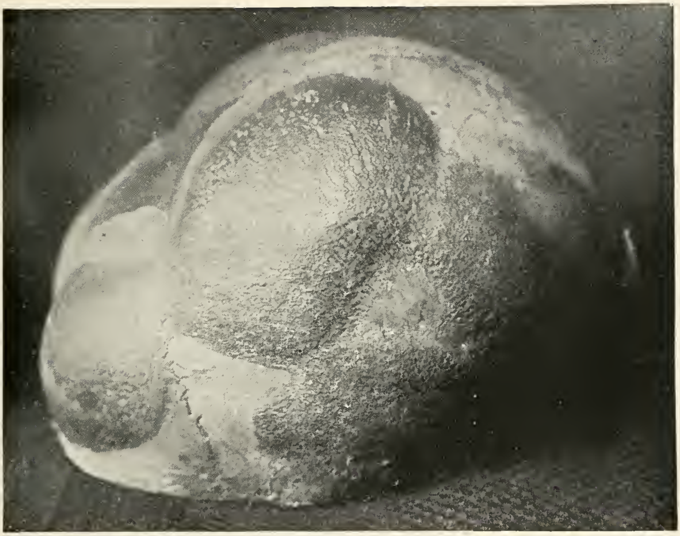
Before leaving thoracic deformities we must mention the effects which they produce on the contained organs. Corresponding to the lines of beads on both sides there is a tract of collapsed lung caused by the direct pressure of these parts of the ribs, while anteriorly filling up the arched space bounded by the sternum and cartilages there is a condition of compensatory emphysema. These changes, as pointed out by Jenner, are constant in the rickety thorax; but extensive collapse, especially of the lower and posterior regions, is very common in fatal cases as the result of bronchitis, to which, indeed, the shape of the chest is contributory. The altered shape of the chest is also responsible for the fact that the apex-beat of the heart impinges to the left of its normal position, and that a white patch is found on the front of the left ventricle, produced by attrition against a beaded rib, instead of on the right ventricle,—the usual situation in the adult heart.

Examination of a vertical section of the *vertebræ* shows a bluish proliferating zone between the disks and the body, also that the body cuts very easily, being softer, more vascular, and of looser texture than in health. The rickety *pelvis* in childhood is more often triangular than oval, but the shape is largely determined by the position the child has assumed during the active stage of the disease.

The Cranium.—We have already mentioned the clinical features of the following lesions: 1. The delay of ossification which is found in the fœtal forms of rickets, extending over large tracts, especially in our experience in the occipito-parietal regions, while even in ordinary cases such delay obtains in the neighborhood of the fontanels and sutures. In these cases membrane is present instead of bone, and no further description is required. 2. The *atrophic forms*, exemplified by tracts of parchment-like yielding bone, and by small pits resulting from extreme thinning of previously-formed bone. These "craniotabes" lesions can be best exposed by removing the calvaria, freed from dura mater. They are then seen to be shallow conical pits, formed by a scooping out of the inner table of the skull-cap; they vary from a slight depression to a pit which has the thinnest possible lamina of the outer table remaining, while in some extreme cases the pit is converted into a hole bounded only by the pericranium. The bone in the neighborhood often yields like stiff parchment, but there is often present a thin deposit of fine red granular osteoid material. It may usually be noticed that the process begins in the centre of the hollows corresponding to the convolutions. 3. *The hyperplastic form.* The hyperostoses, or cranial bosses, the clinical features of which have been described, are seen on section to involve chiefly the outer table, from which they seem to be outgrowths; they consist of red, very vascular, spongioid material, which can be indented with the



RICKETY CRANIUM, seen from above, showing frontal and parietal bosses (porous, vascular osteophytes). Also shows the large anterior fontanel



RICKETY CRANIUM.—Showing frontal and parietal bosses. The porous, vascular character of the osteophytes is shown especially in the upper parietal boss. Parietal eminence not affected with rickety change.

finger or easily cut with the knife. These osteophytes may undergo absorption, or in process of organization become converted into a light, porous, granular bone. In progressive cases they are extended into a diffuse lamina, covering the greater part of the outer surface of the bone, and leading to much massive thickening of the same.

The basis cranii is much less frequently attacked than the vault of the skull, but in rare cases of the limeless-bone form of rickets it is swollen, soft, and can be easily cut by the knife.

Histology.—To understand the histological changes found in rickets it is necessary to have a conception, as clear as possible, of those met with in the development and growth of healthy bone.

Normally ossification occurs in two ways: in the one, bone is formed in connective tissue; in the other, in cartilage. The tabular bones of the roof of the skull are formed entirely after the first or intramembranous method; the long bones, after both methods,—namely, the intramembranous and the intercartilaginous. As most of the changes in rickets can be studied in one of the long bones, as a rib or the radius, it is convenient to restrict our attention to the normal ossification of a long bone.

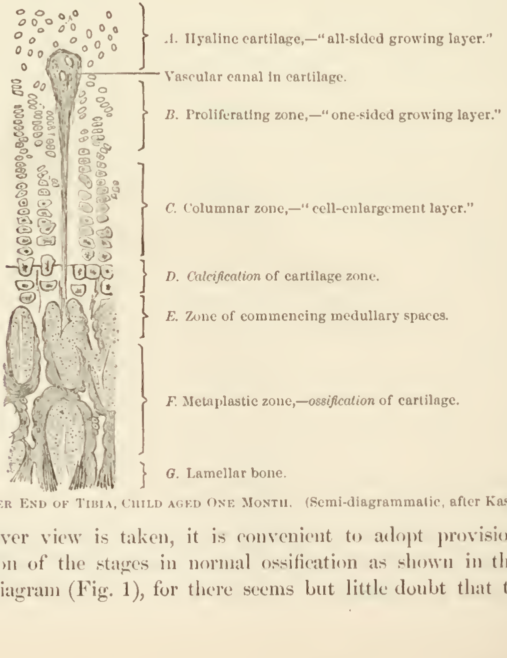
If we examine the phalanx of a very young fœtus just before ossification has commenced, it is seen to consist of cartilage-cells embedded in clear matrix and surrounded by a thin membrane,—the perichondrium.

The cartilage-cells become enlarged in the middle part of the bone (where ossification always begins in a long bone), and besides being enlarged are also flattened and piled up in columns. Next, the matrix becomes hardened by a deposit of calcareous matter which extends between and around the groups of cells, and the spaces enclosed by it containing cartilage-cells are called "primary areolæ." At the same time as this calcification of cartilage, a layer of bone is becoming formed beneath the periosteum by means of the layer containing cells and fibrils on its inner surface,—that is, bone here is true membrane-bone. The next thing to happen is an irruption of the subperiosteal vascular tissue into the middle of the cartilage, one or more apertures being excavated by absorption in the newly-deposited osseous lamellæ and the tissue in question passing through these and burrowing into the cartilage. Here, according to most authorities, it is said to absorb a great part of the calcified matrix, and thus to form large spaces which are filled by embryonic marrow, consisting of ramified cells and osteoblasts, the cartilage-cells disappearing before it,—being either, according to some observers, removed by absorption or, according to others, converted into osteoblasts. All the middle of the calcified temporary cartilage becomes thus excavated into large spaces and replaced by the vascular osteoblastic tissue.

As the calcification of the cartilage-matrix extends towards the ends of the shaft, the osteoblastic tissue closely follows, and, after supplanting the cartilage-cells in the primary areolæ, absorbs parts of their walls so as to throw two or more together to form medullary spaces, or the so-called secondary areolæ: in this way a great part of the primary bone is at once removed. At a short distance below the advancing ossification, the medullary spaces become at first somewhat more enlarged by further absorption, but at the same time their walls (at first formed only by calcified cartilage-matrix) begin to be thickened by the deposition of layers of new bone. The lacunæ containing bone-corpuscles first appear in this deposit; as layer after layer is deposited upon the walls of the medullary spaces, the latter become gradually narrowed into intercommunicating channels which contain little more than a blood-vessel and some jelly-like embryonic connective tissue, with a few osteoblasts applied to the bone.

In the above description it is seen that the calcification of the cartilage is but a temporary arrangement,—a mere scaffolding which is gradually removed and replaced by true lamellar bone,—and no allusion is made to the possibility of the calcified cartilaginous matrix being transformed into true bone. A few years ago Kassowitz revived one of the oldest ideas with regard to ossification,—namely, that the cartilage itself undergoes not merely calcification, but also ossification. To this process which is interposed between cartilage calcification and lamellar bone formation he gives the term metaplastic ossification. For the powerful arguments Kassowitz urges in support of this view, which must still be considered *sub judice*, we refer to his memoir.

Fig. 1.


A. Hyaline cartilage,—"all-sided growing layer."
Vascular canal in cartilage.
B. Proliferating zone,—"one-sided growing layer."
C. Columnar zone,—"cell-enlargement layer."
D. *Calcification* of cartilage zone.
E. Zone of commencing medullary spaces.
F. Metaplastic zone,—*ossification* of cartilage.
G. Lamellar bone.

Lower End of Tibia, Child aged One Month. (Semi-diagrammatic, after Kassowitz.)

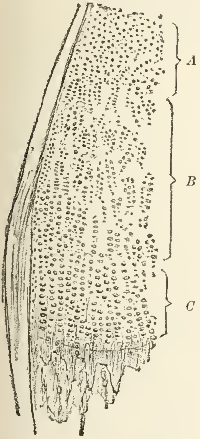
Whatever view is taken, it is convenient to adopt provisionally his classification of the stages in normal ossification as shown in the accompanying diagram (Fig. 1), for there seems but little doubt that the direct conversion of cartilage into bone occurs in rickets as well as in ossifying enchondromata.

Ossification beneath the periosteum also, according to Kassowitz, occurs in two ways. In the first osteogenic fibres are formed in the proliferating layer under the periosteum, and around them bone is deposited; by the union of these bony spicules a reticular tissue is formed, the star-shaped spaces left becoming eventually bone-corpuscles. Later there is a lamellar formation in the medullary spaces, just as in endochondrial-formed bone. This lamellar condition of the bone—both endo- and perichondrial—does not occur till some months after birth; later still, most of the endochondrial bone is removed by absorption, and the bone grows by lamellar deposits beneath the periosteum.

Rickets.—Turning to the microscopic appearances of rickets, we shall find a modification of each of the stages of normal ossification, and first of all it is important to point out that the swelling at the junction of the epiphysis with the shaft (as, for example, the bead on the rib) is mainly composed of cartilage, being principally made up of cartilage-cells much increased in size and number.

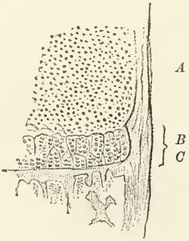
Rickety Changes in Cartilage are manifest only in the two lower zones, namely, the proliferating and the columnar. It is first to be noticed that the relative depth of these two zones varies in size according to age: thus, before birth, when the cartilage is actively growing, *B* is deeper than *C* (see Figs. 2 and 3), but after birth *C* is deeper than *B*. The total depth of the two is smaller the older the child: thus, at the age of three years the depth measures about one-forty-eighth of an inch, but in a six-months fœtus is six times this,—viz., one-eighth of an inch. An accurate knowledge of the depth of this layer of active growing cartilage-cells is essential before rickets can be diagnosed at an early stage, and in a doubtful case the specimen should be compared with a healthy one from a child of the same age. In *fœtal rickets*

Fig. 2.



Rib at Birth.—Letters, as in Fig. 1, note great depth of *B*, the zone of proliferation.

Fig. 3.



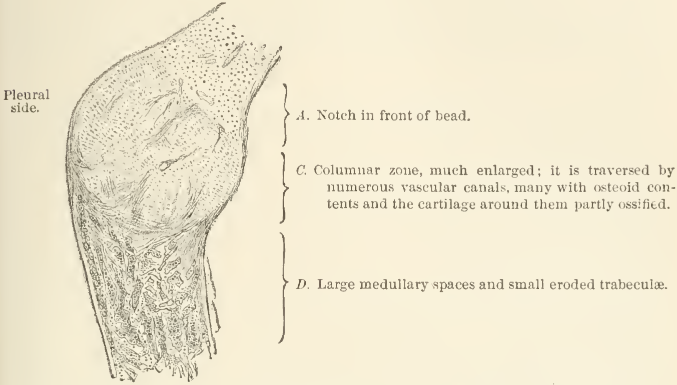
Rib of Child aged Five Years.—Showing shallowness of zones *B* and *C* as compared with an earlier age.

the changes are mainly in the zone of proliferation: each group contains an increased number of cells,—from twenty to thirty,—which are so thickly pressed together that the matrix between them, and between the groups also, almost completely disappears. In consequence of this increased cell-production—the soft cell-contents replacing the matrix—the cartilage becomes softer and its resistance lessened, it has a gelatinous consistence, and the connection between epiphysis and diaphysis is thus loosened. Under the microscope one sees (1) a sinking in of the small-celled zone into the proliferating zone, and (2) a beading of the rib at this spot with the concavity to the pleural side; this notch must not be confused with the one occurring after birth, which is on the opposite or external side. At birth the energy of cell-division lessens, and the already-formed cells and the columns enlarge, and just as fœtal rickets is an exaggeration of the normal process so is post-natal rickets, and the columnar zone may become greatly enlarged while layer (B) is scarcely changed. Thus, instead of being about one-sixteenth of an inch deep at birth, the columnar zone may in advanced rickets measure one-fourth of an inch, and at the age of two years, instead of being as in health about one-fortieth of an inch thick, it may reach one-fifth of an inch. In moderate cases of rickets the enlargement is much less. The matrix between the columns also increases in width, and hence has much to do with the bulging,—the beading of the rib. But this exaggerated growth of the cartilage-cells does not entirely explain high degrees of rickets, for if merely an excessive growth we should expect the greatest breadth to be at the lowest part, whereas it exists at the middle, for the columns do not merely diverge as they descend, but some of the peripheral ones are arched downward and outward. This points to a passive compression, and its explanation is as follows: the hyaline cartilage above and the bone below are not growing at anything like the same rate as the cell-columns, and through the constant addition of new layers of enlarged cartilage-cells there must arise a very great growth-pressure, and as the consistence of the cartilage in its lower zones is diminished there will be compression in the longitudinal axis, and thus an arching of some of its columns and a bulging up at the side above the lower level of the first zone, and often a notch is produced which in post-natal rickets has always its concavity directed out, whereas, as we have seen, in fœtal rickets, before breathing has begun, the concavity is always directed in. The notch referred to, and felt during life just in *front* of the bead on a rib, is often as distinctive a sign of rickets as the bead itself.

We have already mentioned that the columns in some cases diverge downward, instead of converging as in health towards the growth-centre, and in very high degrees of rickets the principal columns do not reach the zone of medullary formation, but abut high up against the perichondrium.

Changes in the Vessels.—Perhaps the most striking and characteristic change is the *increased formation of vessels*, which is apparent even in slight rickets. Normally articular cartilage is quite free from vessels, and even in such actively-growing cartilage as rib cartilage there are only a few; but in rickets perichondrial vessels enter just above the proliferating zone, and descend in this, and are seen in section as tap-shaped processes (see Fig. 4). In marked rickets, besides being increased in number they are widened, thus reducing the area of the cartilage; also, many are to be seen crossing into the columnar zone from the adjacent perichondrium at various heights; they branch upward and downward and in all directions in this zone, and thus produce a confused net-work. In a still higher degree of rickets the vessels in the cartilage-canals become enormously wide,—from twenty to thirty times greater than normal, even reaching one-fiftieth of an inch in diameter,—and look like hemorrhages or large blood-spaces. When involution of such blood-vessels begins, a formation of *osteoid tissue* takes place in the cartilage-canals. In health the canals and their enclosed vessels disappear when the cartilage is completely developed, but in rickets the morbidly increased stream of blood only slowly diminishes as involution of the vessel progresses; hence reticulated bony tissue is deposited between the vessels, and is most abundant near the limit of ossification, for there the vessel is smallest. In *extreme* rickets, where the vessels are greatly enlarged, there is no formation of osteoid tissue, because its determining moment—viz., involution of vessels—is absent. There are also *changes in the cartilaginous tissue* around the medullary spaces in the cartilage, analogous to those metaplastic ossifying processes which occur around endosteal medullary spaces, and there is the same relation to time, for the ossification-change in the cartilage always precedes the formation of osteoid tissue in the canal, just as metaplastic ossification precedes the formation of lamellar bone.

FIG. 4.



A. Notch in front of bead.

C. Columnar zone, much enlarged; it is traversed by numerous vascular canals, many with osteoid contents and the cartilage around them partly ossified.

D. Large medullary spaces and small eroded trabeculæ.

LONGITUDINAL SECTION OF RIB OF RICKETY CHILD AGED FIFTEEN MONTHS. (About three times natural size.)

The result of these complicated processes is that the columnar zone is traversed by osteoid channels, vertical in longitudinal, radial in transverse sections. Also near the perichondrium the osteoid channels are widened and filled with processes of the soft inner layer of the perichondrium, or also partly with areolar tissue of osteoid character. The soft inner layer of the perichondrium has a great similarity to the contents of the cartilage-canals: it is increased not only by its own growth, but also by the melting away of the peripheral parts of the cartilage, and thus finally is produced a great-meshed areolar osteoid tissue, as in the lower parts of the cartilage-canals.

Calcification of Cartilage.—The next event in normal ossification, after the arrangement of the cells into columns and their final development, is calcification of the matrix, and the deposit of tissue advances in a perfectly regular straight line. The anomalies present in rickets vary according to the severity of the disease. In moderate cases the calcification mounts up at the sides along the perichondrium, and at other points along involuting vascular canals, which run up into the columnar zone, and where the plasma-currents are flowing slowly. At the perichondrial border calcification may run as high as the top of the columnar zone, or even higher. But in severe rickets the zone of calcified cartilage, though irregular, does not creep prematurely alongside the vessels, which are surrounded by an uncalcified matrix; for the vessels are dilated and progressively expanding, and in most advanced rickets, where there is a greatly-increased flow of blood, we find the canals without osteoid contents, and without an edge of metaplastically ossified cartilage. Another hinderance to calcification in advanced cases is found in the rapid growth of the large-celled cartilage layer: the cells all strive to develop, but, owing to the continued proliferation, but few reach a perfect state; the pathologically increased plasma-currents stimulate to proliferation rather than to perfection, and hence there is no opportunity for calcification.

Changes in the Medullary Spaces.—In health we see long tubular spaces advancing upward quite regularly and parallel to one another towards the likewise parallel columns of cartilage-cells, in such a way that each space corresponds to one, two, or three such columns, the walls being formed of calcified cartilage. The tops of these spaces are at the same height, and they communicate only at some distance below.

Now, in *moderate* rickets we have (1) the line of advance altered,—some spaces are higher than others; (2) they do not advance in the direction of the cartilage columns, but irregularly, so that a medullary space which is really not broader than a column may by its irregular, oblique course open up and erode many columns of cells; (3) the spaces communicate at higher levels than normally, and (4) between the spaces there are irregularly-shaped pieces of cartilage, perhaps on one side of the space partly eroded, on the other side still unopened, and thus small pieces of cartilage may be found surrounded by medullary tissue. In *severer* rickets very large eroding medullary spaces are seen which eat away the cartilage without any respect to the direction of its cell columns. In *very high degrees* of rickets the formation of medullary spaces is very difficult to follow, for it takes place no longer in the great-celled cartilaginous tissue, but in a tissue traversed by innumerable vascular canals and with but little cartilage in it, and it is very difficult to say which is an endochondrial and which a periosteal vascular canal, for the medullary tissue assumes the character of granulation-tissue. In such cases the zones of medullary formation and of metaplastic ossification are completely abolished, and the spongy bone borders on the large-celled cartilage. We come next to the

Anomalies of Bone-Formation in Cartilage.—In rickets, at all events, there can be no doubt that cartilage is directly changed into true bone: in stained sections red seams of bone are seen not only at the sides of the spaces as in health, but also above and below them; this is usually owing to the very irregular direction of the spaces, but even when normally directed upward sometimes metaplastic bone is seen at the top. Also where in health we see broad unbroken tracts of cartilage we find in rickets *nodules of true bone in the midst of still-unchanged cartilage matrix.* It is especially characteristic of high degrees of rickets to see apparently quite isolated cartilage-cell cavities containing calcified bone with bone-corpuscles, but there is generally some connection with already-formed bone.

The *lamellar bone,* which we have seen in health to be deposited in layers, goes on very rapidly in rickets and may even outstrip the so-called metaplastic, so that we find that medullary spaces which have penetrated into cartilage are already lined with lamellar bone before their walls have undergone metaplastic ossification.

The two prominent *changes in the spongy bone* are—first, distention of existing vessels and a formation of new ones, and, secondly, an abnormally increased erosion of the bony trabeculæ. In health, around each blood-vessel in the spaces there is a good deal of medullary or rich cellular tissue; in rickets, owing to the large size of the vessels, this tissue is reduced to small seams or islets, and so, as the plasma-stream is nearer the bone, new bone is less readily deposited; indeed, the old bone is rather eaten away and the bony septa get smaller, and often two medullary spaces open into each other through the melting away of the intervening septum, and so in severe cases we have very large spaces and narrowed septa, and hence the strength of the bone is much reduced, and small fractures easily occur. The formation of new blood-vessels and the irregular deposit of new bone around them make a complete change in the architecture of the bone.

Perhaps the best-known phenomenon of rickety bone is the *defective or absent calcification of certain parts.* After the eating away of portions of the trabeculæ new bone is deposited, as already stated, and much of it is deficient in lime. This is best seen in carmine preparations of bone not previously softened by reagents: the bone deficient in lime takes a brilliant red color, and contrasts with the silver-gray of the still-calcified parts. Some authorities say that lime has been abstracted from the fully-formed bone, but Kassowitz holds that there has not been an abstraction of lime, but an arrested deposit, the organic part of bone being laid down without the lime. For, he says, the new uncalcified layers which stain bright red are never found to be continuous with the calcified lamellæ, as would be the case if lime were simply abstracted from previously existing calcified lamellæ. The layers run in different directions, and, if they are subsequently eroded, there may be a highly complicated arrangement of bitten lamellæ, both calcified and uncalcified, forming part of the wall of a medullary space; and he contends that it is impossible to have lime salts removed without at the same time the whole structure of the bone being removed.

Periosteal Changes.—The outer fibrous and the inner or proliferating layer are both thickened. Osteoid deposits are formed, and all transitions, from loose, incomplete osteoid tissue with irregular, open, communicating cell-spaces, up to a nearly normal reticular periosteal bone, may be encountered. Later, when the rickety process ceases and a cure begins, the superficial parts calcify, there is an involution of blood-vessels, and hence the vascular spaces become filled with lamellar systems, either completely or leaving narrow Haversian canals in the centre, and so arise the ivory-like hardness and the eburnations of rickety bones met with in adult life.

The formation of this hard, dense bone is universally stated to be a result of the arrest of the rickety process,—a means of cure whereby the bones are rendered firm again; but in rickets in the lower animals there appears to be a formation of massive bone beneath the periosteum, sometimes very porous, sometimes very dense, even when the disease is actively progressing. It is interesting to compare this condition in animal rickets with the hyperplastic cranial changes met with in human rickets.

The foregoing constitute, we believe, following mainly the teaching of Kassowitz, the essential anatomical characters of rickets. Other lesions sometimes found are either *secondary* to a severe and extensive bone-change, or *associated* and dependent, so far as our present knowledge enables us to judge, on an accidental cachexia.

In the former category we should place the wasting of the subcutaneous tissues and the pale, flabby muscles sometimes found.

In the second category would come the profound anæmia, and the enlargement of the spleen, the liver, the lymphatic glands, and the brain.

Of the above changes enlargement of the *spleen* is the most important. Section of the organ shows simple hyperplasia and absence of iodine reaction. In some cases, as pointed out by Dr. Gee, this splenic cachexia is the vestige of hereditary syphilis, in other cases its cause is not obvious.

Enlargement of the *liver* may occur in the splenic cases and in other cases without enlargement of the spleen, when it appears to us to be probably associated with prolonged gastro-enteric catarrh. Enlargement of the lymphatic glands, as described by Sir William Jenner, occasionally accompanies enlargement of the spleen, but in our experience is usually absent in ordinary cases of rickets.

Hypertrophy of the brain and chronic hydrocephalus have been ascribed to rickets, but we have been unable to trace such a relation. Chronic hydrocephalus certainly occurs quite independently of rickets, and the large head appears to us to be mainly explainable by delayed ossification of the sutures, etc., and by massive thickening of the cranial bones.

Morbid Physiology.—Of the histological changes met with in a rickety bone Kassowitz considers that the earliest and most important alteration is increased vascularization of the tissue in which bone is being formed,—a point we think other writers have failed sufficiently to recognize. Kassowitz says that the degree of the rickety phenomena is always proportionate to the intensity of the changes in the vessels, and that cure or reparation follows and advances with diminution of the hyperæmia and involution of the blood-vessels.

Thus, (1) to the vascular richness of the lower zones of the cartilage and their perichondrium we may attribute the enormous proliferation of cartilage-cells and the altered relations of calcification, varying with the intensity of the disease.

And (2) corresponding to the hyperæmia and increased formation of vessels in the endosteal territory we have irregular and premature formation of medullary spaces, with resulting increased osteoporosis of both spongy and compact bone.

(3) In consequence of the hyperæmia of the periosteal system of vessels, there results not only an increased melting of bone, but also a laying down of a loose spongy structure which in the immediate neighborhood of the vessels contains little or no lime.

(4) Finally, with the cessation of the active process the blood-vessels diminish in size, and around them, as their involution proceeds, a new formation of bone rich in lime salts is deposited, and thus all the spongy tissue becomes hardened by a condensing osteitis.

Kassowitz, who traces all the pathological phenomena of the rickety skeleton back to the diseased processes in the vascular system of the bones, regards rickets as a chronic inflammation which always starts in the bone-forming tissues, but later spreads to the older parts of the skeleton, and frequently also to the neighboring joint-apparatus. He compares the process to such an inflammation as superficial keratitis, or to interstitial inflammation of the liver as produced by phosphorus. He shows that absence of suppuration and absence of pyrexia are no arguments against a process being inflammatory, and also points out that *poverty of lime in the bones*, usually held to be so characteristic of the disease, is also met with to some extent in every inflammatory osteoporosis, where trabeculæ deficient in lime are found in the neighborhood of dilated vessels. Here, obviously, the cause is a local one.

Kassowitz also induced hyperæmia by putting an Esmarch's bandage on the limb of a growing animal for some hours, then removing it, repeating the process every three to five days; and he continued the experiment for a few weeks, when examination showed that the increased flow of blood had not only prevented a deposit of lime, but had also caused a melting away of the already-formed bone; proliferation of cartilage and other changes found in rickets were also observed. Hence he contends that every hyperæmia of bone, whether fluxional or inflammatory, is capable of producing a relative poverty of the inorganic constituents, and so he explains the lime-deficiency of rickety bones by the presence of changes regarded by him as characteristic of inflammation.

Two other hypotheses have been advanced to account for this *poverty of mineral constituents*,—chiefly lime and phosphoric acid,—namely, (A) a want of these bodies in the food supplied, and therefore diminished quantities conveyed to the osseous tissue; (B) the acid theory,—that the normally calcified bones are deprived of their lime by the presence of some acid in the circulation.

With regard to the first hypothesis, numerous experiments have been made, and with varied results. Chossat, in 1842, by giving pigeons food deficient in lime obtained easily breakable bones, but Friedleben, who repeated the experiments, proved that, while fragility of the bones resulted, the characteristic changes of rickets were absent. Voit, however, in 1880, obtained positive results, his accurate description of the bone-changes showing that true rickets was present. Baginsky, too, from his own experiments concluded that a simple elimination of lime from the food will produce rickets, the degree of bone-change, however, being much greater when lactic acid is added to the food deprived of lime.

While freely admitting that a constant deprivation of lime may lead to rickets, we should prefer to attribute the result not directly to the withdrawal of mineral constituents from the food, but indirectly to irritation of the delicate ossifying tissues by the altered nutrient juices. Other considerations also show the untenability of hypothesis (A).

Thus, (1) slight but characteristic rickety changes, such as increased vascularization, proliferation of cartilage, etc., are met with when the new-formed bone is *quite normally calcified.* (2) Krukenberg has shown that the ashes of uncalcified cartilage consist principally of lime: hence marked proliferation of cartilage would be quite impossible without a due supply of lime. (3) In the minor degrees of rickets the calcification of cartilage covers a much greater area than normal. (4) Cow's milk is much richer in lime salts than human milk, and yet is more prolific in rickets; and, whatever the food, it would be impossible sufficiently to decrease the supply of lime to produce the great poverty found in bones severely affected by rickets. (5) Finally, the therapeutic administration of lime preparations is not curative, while without any treatment spontaneous cure may take place although the child continues to eat the same food.

(B) That hard bones subjected for some time to the action of an acid fluid will lose their lime, and hence their rigidity, suggested the second hypothesis,—viz., that the yielding of rickety bones was brought about by the presence of some acid robbing them of their previously precipitated salts; and a consideration of the question suggested the probability that this was lactic acid formed in the stomach as a product of bad digestion and conveyed by the circulation to the bones. Schmidt and others stated that they found lactic acid in the bones affected by osteomalacia, but Virchow always found that the medulla of such bones gave an alkaline reaction; and as to rickets, the existence of lactic acid in the bones has never been demonstrated. Its presence in excess in the system of rickety infants was, however, inferred by its detection in their urine (Marchand, Lehmann, Gorup-Besanez); but no proof was adduced that such excess resulted from an increased absorption of lactic acid from the stomach, and Neubauer even failed to find it in the urine of a case of extreme rickets. A further inference of the presence of lactic acid in rickety bones was thought to be justified when Lehmann and Marchand discovered an increased quantity of phosphate of lime in the urine; other observers, however, failed to obtain this, and Zuelzer in 1883 proved that neither lime nor phosphoric acid is present in excess in the urine of rickets.

Of experiments on animals Heitzmann's are the best known. He gave lactic acid by the mouth and subcutaneously to some of the carnivora, and succeeded in producing true rickets; but the food given was also deficient in lime, and the animals were not only rickety but greatly wasted, and suffered from catarrhal inflammation of the bronchi and alimentary canal. These experiments were unsuccessfully repeated, and Korsakow's more recent ones with dogs were also entirely negative. We cannot, indeed, conceive the possibility of any acid being carried in the blood to dissolve out lime from bone.

The purely chemical theories, then, that have been advanced do not even satisfactorily explain the reduced quantity of inorganic constituents, still less do they directly account for the hyperplasia met with at the growing ends of a rickety bone. This, and not deficiency of lime, is the primary and most important fact in the disease; and we have seen that it is produced in animals by the most diverse experiments. The question naturally arises, Are not the characteristic bone-changes the result of direct irritation, the extreme susceptibility of the young ossifying tissue explaining the readiness with which almost any injurious experiment will start rickets?

We may now conveniently pass to the etiology of human rickets, and, by an analysis of the influences to which the developing organism is exposed, endeavor to obtain at least comparative clearness with regard to the conditions productive of rickets.

Etiology.—*Climate, etc.*—Rickets exists in every quarter of the globe: it is most common in those parts of Europe and North America included in the temperate zone, being especially prevalent in cold and damp countries subject to frequent changes of weather, such as England, Holland, and certain parts of Germany and Austria, of France, and of Northern Italy. It appears to be particularly rare in Greece. While frequent in wet and marshy districts, there is no direct evidence of a relationship with malaria, as suggested by Oppenheim; on the contrary, it is really least common in those parts where malaria is worst.

Rickets becomes rarer the farther south we go, and is at a minimum, as regards both frequency and intensity, in the tropics. It is also rare in northern latitudes, as in Iceland, Scandinavia, etc. It is rare at high elevations, especially when the soil is dry. It is much rarer in British India than in Europe, and especially among the poor natives, who live so much in the open air; and S. Watson has pointed out that it is practically limited to the children of soldiers who live in the damp districts shut up in huts.

It is probable that many of the reports with regard to the relative frequency of rickets in different parts of the world require revision, and that they apply mainly to the more obvious and therefore severer types. The rarity of rickets in tropical regions may be contrasted with the prevalence of disorders of the digestive system, which are usually considered to be such important factors in the production of rickets. The open-air life in those hot regions may be justly suggested as the explanation of the rarity of the disorder; and in this connection it may be noted that, even in villages situated high up, where there is much confinement in miserable habitations we meet with severe cases of rickets.

Frequency, etc.—Rickets is one of the most common of diseases, and is especially frequent in densely-populated cities, such as Prague, London, and Manchester, where an average percentage of thirty among young children attending the hospitals has been given by Ritter, Gee, and Ritchie. In the well-to-do classes the disease may be nearly as frequent as in the poor ones, but the type is usually milder. In Vienna, Kassowitz says, the percentage of cases in children under three years of age never sinks below eighty, and he thinks that the lower figures of other authors may be due to the fact that they have not included slight cases of rib-swelling and of craniotabes.

Our own observations tend in the same direction, and we think that, even if the question of craniotabes be left out and attention be carefully directed to the junction-area of the fifth and sixth ribs, there will be no difficulty in finding at least fifty per cent. of examples of distinctive rickets among children under two years attending the out-patient departments of London and Manchester.

Season.—Kassowitz says that worse cases are seen in Vienna during the winter than during the summer, and that this difference is more noticeable with the poor, who are more shut up in the winter and so breathe worse air than the children of the well-to-do. The out-door life in the summer months tends to cure the mild and improve the severe cases.

Ventilation.—The connection which obviously exists between the prevalence of rickets and an in-door life has already been alluded to. The evidence is indeed abundant, as regards both animal and human rickets, that impure air is one of its most fertile causes, and we think it accounts largely for the geographical distribution of the disease and for its greater frequency among the poor. The children of rich parents are often fed on the most unwholesome diet, but they do not inhabit, in company with several other individuals, a poorly-ventilated apartment where the air is saturated with moisture and badly tainted with the products of organic decomposition. An infant who goes out but little will breathe such an atmosphere day and night, and, however suitable its food, there is no lack of irritants for the delicate tissue of the growing skeleton.

Want of sunlight is also a potent factor in the causation of the disease.

Food.—Unsuitable food during the first year of life has been universally regarded as one of the most important causes of rickets. It is certainly true that babies suckled during the greater part of the first twelve months are less liable to be affected than those brought up by hand, and when the disease occurs in them it is of a less severe type. Overfeeding with starchy foods at this period, too much cow's milk relatively to the age or individuality of the child,—to say nothing of the pernicious odds and ends, such as potatoes, pork, raw fruits, which are often freely administered to infants of the poorer classes,—cannot but tend to irritative dyspepsia, and so indirectly not only to general malnutrition, but also to defective and irregular bone-development. But while admitting that premature weaning, and oversuckling, and unsuitable artificial feeding, all tend to rickets, we cannot but think that their influence has been overstated, to the exclusion of other agents, of which we would lay especial stress on (1) bad air and (2) the insufficient covering of the infant's limbs as illustrated by the process of "shortening" or hardening.

Further, it must be admitted that some breast-fed children become rickety in spite of an abundant supply of mother's milk; and, again, that others, especially the first-born children of healthy young parents, who presumably have a good physique to start with, may be fed artificially, with many physiological indiscretions, and yet grow up absolutely free from rickets.

In this connection the following experiments should be considered. Guérin kept puppies on a meat diet for four or five months, when they showed all the signs of rickets in its most typical form, but other puppies of the same litter, suckled in the usual way, remained in good health. Tripier, in more recent experiments on cats, dogs, and chickens, failed to induce rickets by unsuitable food, although the animals died. Dr. Baxter also failed to induce a true rickets by the administration of starchy food to young animals; but it is clear that in his experiments the marasmus produced was so profound that there was not sufficient assimilation to produce rickets, the animals dying of inanition.

The young animals in the Zoological Gardens, Regent's Park, London, artificially fed, but without milk, have furnished to Mr. Bland Sutton luxuriant examples of rickets in every form, to which they have succumbed.

Since on his suggestion a litter of young lions has been fed on milk and cod-liver oil in addition to meat and bones previously supplied, a striking improvement in nutrition has been obtained, and it would seem that the rickets which formerly was so disastrous when only bones and meat were supplied could by this change of diet be overcome without making any change in other surrounding conditions.

Relation to Digestive Disorders.—In a very large number of cases the obvious signs of rickets appear to be preceded by symptoms of gastro-intestinal catarrh, but it is noteworthy that if vomiting and diarrhœa be severe and prolonged, the result is not rickets, but general marasmus. We have made many post-mortem examinations of atrophied infants, in which evidences of rickets were either quite minute or *nil*, and this seems to us to harmonize with the view we have just stated respecting the unsuccessful experiments in the production of rickets in the lower animals. For it appears requisite for the production of rickets to any obvious extent, that a moderate amount of assimilation must occur.

Relation to Syphilis.—Parrot's view, that rickets is the final outcome of syphilis and that syphilis is the only cause of rickets, is much too absolute, and is abundantly controverted by clinical history and observation, and by the parallel manifestations of animal rickets which occur quite apart from syphilis. But his observations show that there may be a very close and complex relationship between the two conditions. It is remarkable that in a large number of cases of craniotabes and of cranial bosses there are evidences of the existence of congenital syphilis, and, although these cranial lesions occur quite independently of syphilis, and must be considered as truly rickety lesions, it seems possible that they are more readily produced in syphilitic than in non-syphilitic infants. The most reasonable view appears to be that syphilis, besides producing its specific changes which are distinct from rickets, and which for the most part run a definitive course of their own, also acts as a chronic depressant to the nutrition of the infant and *quâ* chronic depressant induces rickets.

Relation to Other Diseases.—It is a matter of common observation that rickets is often apparently initiated during convalescence from an acute illness, such as broncho-pneumonia or one of the exanthemata. It may be, however, that such an acute illness has exaggerated a minor degree of pre-existent rickets.

The question of *antagonism* of certain diseases and diatheses to the production of rickets is of great interest. Marasmus has been already mentioned, and there seems also to be some antagonism between tubercle and rickets Thus, children who suffer early from tuberculosis do not appear to become rickety. Again, children who without showing evidence of actual tuberculosis have what may be called the tubercular build of body appear to be little liable to rickets. In such children the teeth are cut early and the bones grow rapidly and are well shaped. On the other hand, rickets is in itself no protection against tubercle.

Heredity.—There is no conclusive proof that rickets is transmitted from father to son. Even the fact in a given case that a father shows signs of former rickets, and that his son becomes rickety, is compatible with the hypothesis that both may have acquired the disease in infancy as a result of defective hygiene, faulty food, etc. Sir William Jenner is sceptical as to the influence of the father in producing rickets, but most authorities agree that the condition of the health of the mother during pregnancy is an important factor. The later children of a large family are much more liable to be rickety than the earlier, and this is more marked among the poor than among the rich, because the conditions of life constantly tend in them to a lower stratum of health.

Relation to Age and Varieties of Rickets.—*Early Fractures.*—In the ordinary cases of rickets, even green-stick fractures seldom occur early. But there are cases of very early fractures—even occurring *in utero*—which are somewhat difficult to explain. A few of these cases occur in premature still-born fœtuses who are the subjects of intra-uterine syphilis.

The fractures occur just above the junction-area of shaft with epiphysis, and on post-mortem examination in some of the long bones inflammatory softening of this junction-region may be found (gelatiniform transformation of Parrot), or the inflammatory process may have come to an end and eventuated in a layer of calcareous deposit through which the fracture takes place. The appearance on section is quite different from that of rickety bone. There is neither a cushion of proliferated cartilage nor a mass of loosely-calcified spongioid bone.

Moreover, the above-mentioned change in the junction-area is often associated with long, diffuse periosteal nodes, giving rise to hard bony laminæ. In these cases may also be found miliary gummata of the liver and sometimes peritonitis.

But there are other cases in which there is no evidence of syphilis and of which the pathology seems to be different. The fractures may occur in the middle of the shaft as well as at the junction-area, they may be single or multiple, partial or complete, and when complete the crepitus obtained is often very striking for such young bones. Bendings of bones as well as repaired (annular intra-uterine) fractures are sometimes to be found. The beads on the ribs may not be very obvious to external inspection, but they are proved on section to be truly rickety. The ossification of the membrane-bones of the cranium is often delayed. There seems no reason to doubt, from the researches of Lauro and others, that these are true cases of intra-uterine rickets. If the process begins early enough, its evolution seems in many respects to be more rapid than we commonly find in ordinary post-natal rickets; for example, alike in the beads and in the ends of the long bones the broad cushion of proliferating cartilage has been partly replaced by brittle calcified spongioid bone, which is a comparatively late phase of the post-natal rickets. So also with regard to the layer of bone forming the shaft. This explains the undue brittleness of the bones in these cases.

Many of the cases of severe and early intra-uterine rickets with multiple fractures die soon after birth, but other cases, in which probably the process has begun at a later period, show definite signs at birth, and, whilst some of the deformities prove their rickety character by undergoing gradual involution within a few months of birth, typical signs of rickets in other parts of the body develop. For example, a child who was under the care of one of the writers had been born with much defective ossification of the back of the skull, and with considerable bowing of the anterior part of both thighs, which were found to be very tender. Within the first three months of life the bent thighs lost their tenderness and became slowly and definitely straighter, whilst a green-stick fracture appeared in the upper part of one humerus and some suspicion of a green-stick fracture appeared in the corresponding part of the other. Beads on the ribs and grooving of the thorax also became very manifest.

There is probably a descending scale of intra-uterine rickets depending on the period of initiation and the activity of what we may provisionally call the irritant which gives rise to the disease. There are, first, those cases such as Lauro has described, in which the disease begins early and runs what must be considered as compared with post-natal rickets an accelerated course, in which fractures may occur in intra-uterine life or during the act of birth or shortly after birth with the very minimum of violence, and in which it would appear that the premature calcification of spongioid bone is responsible for the excessive brittleness. Secondly, there are cases like that just mentioned in which the process has not progressed so far as in the first case, but in which rickety deformity in some bones exists and in which the premature rickety change in other bones is proved by early green-stick fracture. In these cases the rickets is progressive for a time at least after birth. Thirdly, there are cases, which according to Kassowitz's and Schwarz's investigations are tolerably common, of *slight* intra-uterine rickets. The clinical verification at birth of these cases may require some care to establish, but post-mortem examination proves the existence of rickety lesions, and the frequency with which they are found strongly suggests that such slight intra-uterine rickets may often be the rudiment which develops into common post-natal rickets.

So-called Fœtal Rickets—Fœtal Cretinism—Achondroplasy.—Over and above the cases which we have before described as true intra-uterine rickets, there are several specimens which have been described by different observers and in which the common characters are so striking that they form a distinctive group.

The cases in question are generally still-born, or if they survive it is only for a short time.

There is often a considerable amount of subcutaneous fat marked off on the limbs by curious transverse furrows. The most striking feature in these cases is the disparity between the length of the limbs and that of the trunk. The upper limbs when laid alongside the trunk often do not extend lower than the level of the umbilicus, and the fingers are particularly stunted.

The lower limbs are also stunted. When the limb-bones are removed, they are seen to be firm and smooth, with the normal curves somewhat exaggerated, especially in relation to the length of the shafts. On section the bone of the shaft is found to be unduly compact. There is no proliferating zone of cartilage at the junction-area, and the ossifying centre of the epiphysis when developed is situated not in the centre of the epiphysis, but along the line of junction with the shaft. Microscopic examination shows that the preliminary row formation of cartilage-cells does not take place, or only to a very slight extent, and the whole process of bone-formation in its initial as well as its later stage is derived from the intrusion of periosteum.

It is the failure of the columnar row formation of cartilage-cells which probably determines the arrest or stunting of length-growth of the limb-bones, and it was on this character that Prof. Parrot based the title which he gave to this group of cases of achondroplasy.

It will be obvious that in spite of superficial resemblance these cases have nothing essentially in common with rickets: histologically, indeed, they constitute a group which is the antithesis of rickets. It is interesting to note the difference from a true rickety bead which the rib at its anterior extremity presents in one of these cases. There is a thickening round the end of the costal cartilage, but it is found on section to be a bony investing sheath or ring which is a prolongation derived from the active periosteum of the rib and in which proliferating cartilage plays no part.

The skull-changes are very remarkable. The membrane-formed bones are well developed, but the cartilage-formed portions are stunted. In these cases there exists the curious tribasilar synostosis first described by Virchow. There is premature union of the basi-occipital, basi-sphenoid, and præ-sphenoid, thus forming one short continuous bone, which accounts for a remarkable shortening of the basis cranii.

Eberth has found changes parallel to those above described both in the long bones and in the skull in some specimens of malformed calves, and has given the title of calf-cretinism to the condition. As an alternative name to Prof. Parrot's it would seem reasonable to call these cases fœtal cretinism, though it is doubtful whether the thyroid gland is constantly altered as it appears to have been in Virchow's case.

The So-called Acute Rickets.—The great variation between different cases in respect of number of bone-lesions, mode of onset, and amount of local distress and of constitutional suffering has been recognized by all writers on rickets. As to mode of onset, cases have been divided into those which begin abruptly and those which begin insidiously, and for some writers cases of acute rickets are nothing more than ordinary rickets commencing abruptly.

But careful clinical study will show that there is a group of cases to which the title of acute rickets has been applied which have many characters in common separating them from ordinary rickets. For a full account of these cases the article on Scurvy may be consulted, but it is adequate in the present article to enumerate briefly the striking features. The lower limbs are most severely and characteristically affected; they are tense and shiny and often quite immobile. The tenderness is excessive. At the junction-areas sometimes crepitus can be obtained, and from these regions there is a sheath of cylindrical swelling extending for a varying distance along the shafts.

The bones of the upper limbs may also be affected, and the cranium and ribs. Fractures have been observed in the upper limbs and in the ribs. The anæmia in these cases is profound, and the great majority of them present *more or less sponginess of the gums.* On post-mortem examination of the bones there is found often an extensive subperiosteal hemorrhage, most marked in the limbs, which are tense and immobile. This extravasation is enough to separate the cases from those of ordinary rickets.

Infantile Osteomalacia.—We have already seen that in rickets there are many processes going on side by side,—namely, proliferation of cartilage, the formation of loose spongioid bone which contains lime salts, the absorption of bone, leading to rarefaction of the shaft, and the deposition of new bone *which is almost if not altogether limeless.* The proportion in which these processes are found may vary within wide limits. If the first two are very active, ordinary rickets with its enlarged bone-extremities is the result. If there is much absorption, there is then proneness to partial fractures of the trabeculæ which remain, and also of some of the unsupported cortical layers, and in all probability these fractures take a part in the production of the deformities of the shafts. If, in the third place, after there has been considerable absorption, the deposition of limeless bone both under the periosteum and in the medullary canals becomes the prominent feature, then *rickety osteomalacia* is the result. The bones in these cases are small and elastic and do not readily break. This last variety has in our experience occurred only in very marasmic children, and in the cases reported by Dr. Rehn the same fact was observed. A case has been recorded by one of us which showed remarkable differences from the above variety. A marasmic child, seven and one-half months old when first seen, presented remarkable bendings of all the limb-bones and great flexibility of the cranial bones, but the chest showed neither beads nor grooves. It was stated that the bendings had been present since birth. On post-mortem examination all the long bones were extremely brittle, and on section the shaft was found to consist of a thin cortical shell of apparently normal bone which enclosed soft dark-red pulpy material. On washing this material away a scanty trabecular gritty framework was seen, whilst in some places the bone was completely absorbed, leaving the appearance of cystic cavities. *There was no proliferation of cartilage at the junction-areas, and this was proved microscopically.*

The bones forming the roof of the cranium were thin, and consisted, like the limb-bones, of thin cortex with pulpy contents. Many fractures of the ribs were found near their posterior angles, but no indication whatever of beads at the anterior extremities. Obviously the features of this case agree with those of adult mollities ossium or *true osteomalacia* and differ completely from the cases which we have previously described as belonging to the *limeless-bone* variety of rickets.

In the latter variety proliferation of cartilage is present, and after the absorption of bone there is deposition of new limeless bone which forms a nearly solid flexible shaft, whereas in the case described by one of us proliferation of cartilage is completely absent, and absorption of bone has attained an extreme degree, the trabecular structure being largely replaced by pulpy cellular material.

Late Rickets.—The onset of active rickets at periods later than two years old is certainly rare. Sir William Jenner refers to the case of a boy in whom the symptoms of the constitutional disease did not manifest themselves till he was a little more than three years old, and to a girl aged nine years who was then only beginning to suffer. Other observers have stated that rickets may commence in adolescence, but we do not know of any post-mortem evidence on these cases.

Our own experience is confined to two cases in which there were very active manifestations at the age of eleven years, proved post mortem to be truly rickety, presenting characteristic exuberant changes at the junction-area, under the periosteum, and in the medullary portions of the long bones, with typical fractures. But in one case certainly, and in the other case probably, there had been infantile rickets which had subsided. The active symptoms at the age of eleven years were regarded by us as remarkable recrudescences rather than new developments of the disease.

Pathological Summary.—A review of the facts disclosed by a study of the morbid anatomy and etiology leads us to the following conclusions:

1. That just as, clinically, enlargement of the junction-area is the most characteristic sign, so its anatomical equivalent—namely, proliferation of cartilage, with the associated increased vascularization—must be regarded as pathognomonic of rickets, softening of bone being a more variable and less distinctive feature.

2. That histologically it is impossible to distinguish such a condition from that met with in the early stages of an ordinary inflammation. It is obviously something more than a mere chemical change: such overgrowth could result only from direct stimulation of the tissue itself. Between a simple hyperplasia and an inflammation there are, no doubt, many connecting anatomical links; and while the theoretical question, whether rickets is to be classed as one of the links in the chain, or as a *bona fide* inflammation, is not easily settled, the plain and practical outcome of a study of these changes is, surely, that we have to deal with *an irritative overgrowth of the osteogenetic tissues, and that this, and not deprivation of lime, is the primary fact in the disease.*

3. Whatever the irritant causing the overgrowth described,—whether a single substance, as lactic acid, or any one of several substances,—it is quite certain that it is easily developed in infant life. For if one fact stands out more prominently than another in the pathology of rickets, it is this, that almost any injurious influence brought to bear on a child during the period of most active growth tends to produce rickets: a chill to the surface, the inhalation of noxious gases, the assimilation of ill-digested fluids, the syphilitic virus, etc., may each develop some irritant in the blood which, however mild, easily acts on the tender walls of the young vessels in the growing parts of a bone; in health a new formation of vessels is going on there, and a physiological increase is easily stimulated into a pathological one. It seems probable that if the irritant acts suddenly and profoundly, so as to interfere in a marked degree with assimilation, atrophy and not rickets is induced.

4. There is sufficient evidence that in many cases rickets is initiated at a very early period,—namely, during the last three months of fœtal life or during the first few months after birth, when, as Kassowitz points out, the bones are most actively growing. There is also a correspondence between the age at which spontaneous cure occurs in a particular bone and the time when the energy of growth of that bone is diminishing. The rare examples of "late rickets" are probably always caused by irritation of lesions which were initiated very early in life and have remained latent.

Course and Prognosis.—In this disease different portions of the skeleton become progressively involved, probably in relation to their respective periods of developmental activity, and, as there is a natural tendency to involution of the bone-lesions, the disease may be subsiding in one region when it is only starting in another: thus the cranial changes may be approaching the end of their cycle when active epiphysial changes are only commencing. It seems possible that the cranial lesions may pass through their evolution with a minimum of involvement of other parts of the skeleton. The same may be said of slight beading of ribs, which may appear, remain for a time, and then pass away without any other outward manifestation whatever. It is, however, always important to remember that slight changes may be present elsewhere which give no clinical indication. Further, in some cases the incidence of the disease in one region may be very pronounced and the lesions in other regions slight.

The time occupied in the complete evolution of the disease varies within wide limits. When rickets is an *early* fœtal disease, its progress is probably much more rapid than that of the ordinary form of rickets, with which we are acquainted. But in the ordinary form there are great variations, dependent to some extent on the degree to which existing injurious influences are removed and on the liability of exposure to fresh sources of irritation. Definite exacerbations and periods of latency may be observed. This is probably the case in late rickets.

As striking examples of complete involution we may mention the entire disappearance of the beads on the ribs and the vertical grooves on the thorax, which are never seen in the adult, whilst the pigeon-breasted chest, which may arise independently of rickets, often persists in adult life.

Curvatures of the limbs tend to right themselves with further growth of the bones after the rickety process has come to an end in that region, but we can never foretell what degree of straightening will take place.

Arrest of growth with resulting dwarfing in length of the limb-bones is a common result of severe rickets, and may coexist with a fair development of head and trunk.

Prognosis.—Rickets, though not in itself a fatal disease, is liable to many serious complications, and, if severe, by impairing mobility interferes with proper nutrition: in slighter cases, on the other hand, the general health seems to be scarcely affected. The serious complications are those of the respiratory tract mainly, in consequence of the thoracic deformity which contributes to pulmonary collapse. Bronchitis, broncho-pneumonia, measles, whooping-cough, and laryngitis are badly borne by the rickety infant.

Laryngismus stridulus and convulsions are occasionally fatal.

The supervention of splenic anæmia, although not necessarily fatal, is serious even in a slight case of rickets.

Treatment.—*Prophylaxis.*—Adequately considered, this involves the complete hygiene of infancy and early childhood, and for a full discussion of the subject we refer the reader to the several articles dealing with it in the preceding volume. But it is necessary to state some of the most important points bearing on the care of infancy, the more so that the measures best adapted for the prevention of rickets constitute the essential part of the treatment of the disease when present.

The Pregnant Mother.—Accepting Kassowitz's teaching that many cases of rickets begin in intra-uterine life, it is obvious that we should consider the maintenance of the mother's health at the maximum during pregnancy as of the first importance. Although we are ignorant of the cause of the anomalies which sometimes occur,—*e.g.*, the pregnant mother in fair average health and the new-born infant poorly nourished, and *vice versa,*—yet, in general terms, the maintenance of the mother's nutrition is equivalent to giving the infant a good start. Two conditions in the mother seem pretty clearly to determine the occurrence of rickets: first, repeated child-bearing *per se*, and, second, the continuance of suckling during pregnancy.

The Food of the Infant.—The great desideratum is to maintain the mother's milk in respect of quantity and quality.

In young mothers we hold that suckling may be often continued as the exclusive mode of feeding for ten months with advantage to the infant and without injury to the mother. The practice among the well-to-do of weaning the child on account of slight failure of the mother's nutrition is mischievous and short-sighted. To our great surprise, and in some cases against our urgent advice, we have seen mothers continue suckling through febrile diseases, without obviously interfering with the nutrition of their infants, and also themselves making good recovery from these affections. We have also found, in some cases where there was considerable debility and some anæmia in the early period of lactation, that these conditions have manifestly improved after the suckling has been continued for a time. With respect to the occurrence of the catamenia as an indication that suckling should be suspended either temporarily or permanently, although we have seen during such times some disturbance in the infant's digestion set up in the way of loose stools or even vomiting, such disturbance has in our experience been slight, and we consider that it is unwise on this ground to discontinue suckling.

There are undoubtedly rare cases in which suckling has been followed by convulsions in the infant, and there are not a few in which on exclusive suckling the infant does not thrive and in which after being placed to the breast it is dissatisfied; but even in these cases we believe that partial suckling ought to be attempted. The alleged danger of mixing the milk —that is, of combined natural and artificial feeding—is entirely illusory.

With respect to the food of the nursing mother, it need only be here stated that over and above a mixed nutritious diet there ought to be a fair amount of fluid, and that common experience teaches that milk gruel, cocoa, and cod-liver oil may often be assimilated with much advantage during lactation by those who at other periods would be unable to digest them.

The infant ought to be fed at first every two, then every three hours. Without laying down the absolute rule that in no case ought a shorter interval than two hours to elapse, it ought to be stated that before giving the breast to a crying infant it should first be ascertained if the distress is caused by flatulence with a tight binder or by cold extremities.

Artificial Feeding.—If the mother's milk fails, whether partially or entirely, it becomes necessary to supplement or replace it either by a wet-nurse or by some form of artificial feeding. The first of these alternatives, although doubtless the simpler solution of the two, ought not to be insisted on before a reasonable attempt has been made to bring up the child by hand.

The most convenient adjunct to the mother's milk is scalded diluted cow's milk. It ought to be scalded (sterilized) in order to destroy disease-germs, and especially in the summer-time, in order to prevent the lactic-acid fermentation. It ought to be diluted chiefly because of the excessive amount of casein contained in cow's milk as compared with human milk. As to the amount of dilution no absolute rule can be given; it varies with the richness of the milk and the digestive capacity of the infant. The common formula—equal parts of cow's milk and water during the first three months, and gradual increase of the amount of cow's milk up to full strength at twelve months—is in many cases satisfactory, but the amount of the dilution must obviously vary with the richness of the milk.

The true test, however, is to be found by examining the condition of the child's stools. If they continue of a bright gamboge-yellow color, of a soft consistency, not markedly offensive, and in number not greater than two in twenty-four hours, the result of the feeding is so far satisfactory. If white masses of unaltered casein appear, some alteration should be made. Simple dilution may be adequate, or the substitution of freshly-prepared barley-water as the diluent instead of simple water. This simple decoction, besides being in itself nutritious, acts beneficially by favoring the precipitation of the casein in smaller masses than would otherwise occur.

The addition of a small quantity of long-boiled and strained gruel to the diluted milk serves the same purpose, and so does the use of isinglass.

Lime-water has been a time-honored diluent for cow's milk, and, on account of an exploded pathology of rickets, has been held in high repute; but the amount of lime held in solution in ordinary lime-water is so minute as to be of little value, and if an appreciable amount of the alkali is desired, saccharated liquor calcis should be used. The writers believe that barley-water in the majority of cases is quite adequate and that lime-water is unnecessary.

If the casein is still a difficulty, one of five courses may be adopted:

(1) The milk may be "humanized." The cream having been separated, some of the casein is precipitated by curdling. To the whey still containing a little casein the cream is now restored, the resultant being a nearer approach to the mother's milk than before.

(2) Or, to the freely-diluted cow's milk or to freshly-prepared whey, cream may be added in the proportion at first of a teaspoonful of cream to about four ounces of diluted milk or whey, the cream being gradually increased.

(3) Another plan is partly to peptonize some of the casein of the milk by the use of some one of the digestive ferments. Of these, pancreatic ferment has lately come much into vogue, and is certainly very convenient. But, though sometimes useful, this plan is probably less satisfactory in its result than (1) and (2).

It is better to call into play the functions of the glands of the stomach in the natural digestion of a smaller amount of casein than to present to the stomach an already partly-digested pabulum.

(4) Condensed milk is undoubtedly tolerated in some cases in which fresh cow's milk utterly fails. It is useful as a temporary expedient, and is valuable on board ship or in travelling, when the quality of cow's milk cannot be relied upon, but it is not to be trusted for lengthened periods except as an adjunct to mother's milk or in combination with fresh cow's milk. (See article on Scurvy.)

(5) Cow's milk may be put aside altogether and either ass's milk or goat's milk employed, in both of which the proportion of casein is much less than in cow's milk. Of the two, ass's milk is generally the more successful, and it may be given undiluted, though it ought always to be scalded except where satisfactory daily inspection of the udder of the ass can be obtained.

Some reference must be made to the various artificial infant foods which with or without the imprimatur of Liebig's name have flooded the English and American markets. Of the *non-malted* foods and those which are recommended to be prepared with water only, scarcely anything need be said except in condemnation. As temporary expedients during severe gastric catarrh they may be useful, but they are unfit for continued employment as food for infants. (See article on Infant Scurvy.)

Of the malted foods, probably all the chief varieties are useful to some extent, but they also are inadequate when given alone, for, according to the careful investigations of Dr. Cheadle, they come short of being complete foods because of their deficiency in fat and in proteid.

The writers believe that their chief value in early infancy, when given in small quantity with milk, consists in their facilitating the breaking up of the casein-coagulum into small and manageable masses. When the infant has passed the age of seven months, the quantity of farinaceous material added to the milk may be cautiously increased. Of such material we know nothing better than oatmeal boiled for three or four hours and then strained, the strained product being added to the milk. Plain biscuit or rusk, if boiled and sieved, may be added to the milk during the last quarter of the first year. Towards the end of the first year, if the teething have proceeded satisfactorily, small pieces of rusk with fresh butter or bacon fat may be given to the child to chew.

Between one year and eighteen months thoroughly-mashed potatoes with gravy may be given, and indeed before this time some potato may often be given in milk with advantage if thoroughly boiled and sieved. Also between one year and eighteen months some light pudding, with milk, may be commenced, and likewise a boiled egg and a little broth. The writers believe that, as a rule, it is best not to begin the use of solid meat until after the age of two years, for, although it is doubtless in many cases well digested, its employment tends to put into the background the child's liking for and dependence on milk and the various cereals. The writers believe that carefully-selected soft succulent fruits, such as the juice of oranges, morsels of baked or grated raw apple, and well-cooked vegetables, may with much advantage be given to children of eighteen months and even younger. We should, however, avoid the administration of preserved fruit and jams, which often induce acid fermentation, and in like manner stringy vegetables, which are apt to set up in young children dysenteric diarrhœa, should at this age be forbidden.

Clothing.—In the damp, cold, and variable climates of England and America, in general terms, the safest clothing for infancy and childhood consists of warm, close-fitting, equable woollen under-garments, the thickness of which should vary with the season, whilst the upper garments should be loose and light, but altered in thickness and number according to the out-door temperature.

We hold that every child under two years of age should whilst in the outside air be clothed in such a way that arms, legs, and neck are uniformly covered with woollen fabrics, and that in-doors during cold weather the same rule should be adopted. The process of "hardening" by curtailing the amount of woollen under-clothing, so as to leave the neck, part of the legs, and arms bare, is surely mischievous, and stout, vigorous children are strong, not in consequence of this plan, but in spite of it. The best methods of "hardening"—in other words, of increasing the resistance of skin and mucous membranes to the reflex disturbances set up by cold and damp—consist, we believe, in careful ventilation, in obtaining for the child as much out-door life as possible, and in the discriminating use of baths and of friction to the surface of the body. With respect to ventilation, the average mother and nurse have still to learn that it is necessary to avoid differences in temperature between the nursery and the rest of the house, and between the house and the outside air. If the temperature of the nursery in winter be kept above 60° F., chills on going into other rooms and especially on going outside are almost inevitable. Of all modes of ventilating nurseries, we believe that open fires give the greatest safety, as it is then possible to have some simple and *constant* aperture communicating with the external air.[1]

The advantage of out-door life is now frankly recognized with respect to healthy children, but the value of some free exposure to air for tender and susceptible children has scarcely filtered into the lay mind as yet. It is certain that many delicate children may with great advantage be taken out of doors for *very short periods*, at tolerably frequent intervals, if care be taken not only as to clothing and body-warmth but also as to the timely administration of food shortly before they are taken out.

With respect to *baths*, we think the temperature ought not to be above 90° F., and after six months the morning bath may be reduced to 80° F., and at twelve months to 70° F. Without fixing any absolute time, we believe that the duration of the bath given to children is often too long. After six months of age great advantage is derived by squeezing a large spongeful of water down the back before taking the child out of the bath. This ought at first to be of the same temperature as that of the water in the bath, and the child ought to become accustomed to this without being frightened. By slow degrees the final spongeful of water applied in this way ought to be made colder, and if carefully done the bracing and hardening effect of this simple measure will become very manifest. The bath should always be given before the fire, and followed by vigorous rubbing.

The liability to cold extremities which corresponds with the feeble peripheral circulation of childhood, and which sometimes persists through life, ought, we believe, to be most zealously watched and neutralized. Whenever hands and feet are found blue and cold, they ought to be rubbed until they are warm. By so simple a method, that which is liable to become a pathological habit may certainly be controlled. Some children get blue extremities after a bath whether it be warm or cold. An excellent method in such cases is to precede the bath by vigorous rubbing, either of the whole body or of the extremities, with oil, then to soap the body freely and give the bath as quickly as possible. In these cases the final affusion with water slightly colder than that of the bath is often of great value. The dry rubbing after the bath ought to be very thorough and prolonged.

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[1] From 65° to 70° F. is a better temperature for the nursery during the cold winters of the larger cities of the United States. Our methods of heating houses, though not the most healthful, give the entire house the same degree of temperature.—EDITOR.

General Treatment of Rickets.—Having ascertained the existence of active rickets, however slight, we must next investigate the causes which may be supposed to give rise to the disease. These may be manifold, and it is often difficult to estimate the exact share of each factor, but in every case it is our first duty to rectify, as far as may be, any departure from the general lines laid down in the preceding section: thus, the feeding and clothing of the child, the ventilation of its room, its bathing, and the amount of its out-door life, should each be minutely regulated; for the question of hygiene in its widest sense dominates the treatment of rickets, and, when placed on a right basis, the natural tendency of the disease to recovery is largely insured.

So far as we know, there is no specific for the cure of rickets. Yet it is maintained by Kassowitz that phosphorus acts directly and with benefit on the epiphysial lesions. He commences with half-milligramme ($\frac{1}{125}$ of a grain) doses, dissolved in almond or olive oil; and he claims that under this treatment, without modifying hygienic conditions, the bone-lesions and the general nutrition perceptibly improve. We have no personal knowledge of the value of this treatment. But as to the benefit derived from cod-liver oil there is universal agreement. It may be given with confidence in most cases, but ought to be diminished or suspended if it obviously gives rise to vomiting or diarrhœa. The dose ought not, as a rule, to exceed two or three teaspoonfuls daily, even at the age of eighteen months. It may often be given with advantage in five- or ten-drop doses to the youngest infant. It is tolerated in larger quantities in winter than in summer, and is best given after meals or the last thing at night. As a rule, cod-liver oil can be taken by itself, and when this is the case it is far better to give it alone rather than in one of the numerous trade emulsions, the composition of which is uncertain: the simple combination with extract of malt, however, is often useful, especially as leading the way to the administration of the oil pure and simple. When, owing to digestive disturbances, the internal administration of cod-liver oil is impossible, in some cases there appears to be advantage obtained by its inunction into the skin. There is fair evidence of some absorption taking place.

Scarcely inferior in therapeutic value to cod-liver oil is the careful employment of baths and friction of the surface of the body. We have already given in detail, in the prophylaxis, the necessary cautions as to the employment of baths for infancy and early childhood, and we only wish in this section to emphasize the great value, in the treatment of rickets, of the modified douche given after the baths. When frequently used and given very rapidly, this often has a markedly beneficial effect on the head-sweating and on the general nutrition of the skin. Warm salt baths followed by the douche are also valuable.

Friction with some simple oil not only improves the nutrition of the superficial tissues, but also is useful for the development of the flabby muscles, and much can be done in this way in averting spinal and other deformities. When the limbs are tender, shampooing should be suspended or done with the greatest care.

Treatment of the Bone-Lesions.—If there is much tenderness, and if there are green-stick fractures, or acute bendings of bone, it is best to maintain the horizontal position and provide for adequate support and immobility. During early active phases of rickets, constant care should be given to supporting the back. When the tenderness of the limbs has subsided and the child is anxious to stand, if there is any deformity it is wise to err on the side of over-caution, for, although there is a natural tendency to involution and many deformed bones ultimately become straight, we can never be sure that the restoration will be complete.

The value of splints, as usually applied, for the direct purpose of overcoming deformities, may be open to doubt, but a splint applied so as to extend well beyond the foot and thus interfere with *walking* is often very valuable. During enforced rest, if the bone-tenderness has gone, shampooing is doubly indicated.

For the after-treatment of the bony deformities, the reader is referred to the surgical articles of this work.

Of the various tonics useful for rickets, the simple preparations of iron made without syrup seem to be the best.

Treatment of Complications.—Of the complications of rickets, the first to be considered are the gastro-intestinal disturbances, not only because of their general interference with assimilation, but also on account of their tendency to aggravate the rickety bone-change. Here the regulation of diet plays the chief part in the management of the case. No treatment of rickets is satisfactory that does not aim at getting the evacuations of a healthy color and consistence.

With regard to the simple white stools of undigested casein, we consider the administration of mercurials day after day in these cases very bad practice. We have already pointed out, in the prophylaxis, the various methods which seem most useful in dealing with casein-indigestion, but here it must be stated that some children have a remarkable incapacity for the digestion of milk in anything more than the smallest quantities at a time. In such cases it is better to supplement the milk, or indeed entirely replace it, for a day or two, by other food. Here we strongly urge the use of various fresh aliments rather than the immediate recourse to the patent artificial foods. Among such temporary substitutes for milk we place in order barley-water, white of egg and water, cold beef juice, chicken or veal broth. If the stools are very offensive, in addition to the regulation of the diet, certain drugs may be necessary. It is useful to begin with a dose of castor oil, in order to clear the bowel of irritant material. This may be followed for a time by a simple castor-oil mixture, composed of five or ten drops of the oil for each dose, combined with mucilage and some aromatic water. Soda, rhubarb, and bismuth are often useful, and there cannot be a doubt that gray powder is sometimes valuable.

When frothy stools or gaseous distention of the abdomen are marked features, we have seen the greatest benefit from the timely use of simple enemata, and the same may be said when much straining occurs, with the passage of stringy or blood-streaked mucus. In the latter condition it is very important to keep the child in bed, and, if the stools be frequent and copious, the combination of minute doses of opium with castor oil and the use of small starch enemata are, we believe, more valuable than astringents.

The *respiratory complications* come next in importance to those of the alimentary tract. The proneness to bronchitis, collapse, and broncho-pneumonia has been already mentioned, and nothing special need be said in this article as to the treatment of these conditions, except that rickety children bear all depressants very badly.

In the treatment of the *nervous complications*, attention ought first to be paid to the removal of peripheral irritation, and this, we believe, is most often found in the alimentary tract.

It is interesting to note that many of the suggestions which have proved most useful for laryngismus have been based on the improvement of the general tone,—viz., cod-liver oil, cold sponging, and frequent exposure to fresh air. In convulsions and tetany the same principles hold good,—viz., that the removal of irritants (chiefly intestinal) and the use of cod-liver oil and tonic treatment generally are more valuable than sedatives.

SCURVY.

By THOMAS BARLOW, M.D., F.R.C.P.

Scurvy is a disease which in adults is characterized by great anæmia, sallow muddy complexion, extreme debility and proneness to syncope, sponginess of gums, and ecchymoses in various parts of the body, but especially in the lower limbs, in which, also, brawny indurations occur. It has a definite relation to the deprivation of fresh vegetables, and is almost immediately ameliorated by their administration, but appears to be controlled also by fresh raw meat and by fresh milk.

The object of this article is to show how far this disease, as it occurs (1) in childhood and (2) in infancy, agrees with and differs from the adult type.

For purposes of definition, infancy is considered to extend from birth to the age of two years, and childhood from two years to ten years.

GROUP I.—ILLUSTRATIVE CASES IN CHILDHOOD.

Case A.—Elizabeth O., aged ten years.[1] Had been a bottle-fed child. Had had measles, whooping-cough, and scarlet fever, though without obvious sequelæ. Always extremely fanciful about her food. The family, though poor, had meat once or twice weekly and vegetables daily, but this child would take little but bread and butter; "meat and vegetables she would not touch, and milk she did not like."

For the last four summers she had lost strength in her lower limbs, and had suffered much pain referred to her knees and ankles. On several occasions, it is said, the knees and ankles have been swollen and tender, but they have never been hot.

At the same periods the child's gums have become swollen. Sometimes the swelling would occur very suddenly in the night, and would on some occasions subside after a few days. Blood has oozed from them at times, and the smell has been very offensive. She has bled a few times from the nose, but no blotches have appeared on the skin.

The child has also been subject during the last four years to what are called fainting-fits. These were specially apt to occur at the time of the joint-symptoms. In these attacks she would remain perfectly still for an hour, with her eyes open and her teeth clinched, but she was not said to be paler than usual on these occasions.

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[1] I have to thank Dr. W. H. Dickinson for his permission to record the notes of this case, which I made when the child was under his care at the Children's Hospital, Great Ormond Street, and subsequently when under my own care at the Convalescent Department.

During the winters she was better than during the summers, and she was then able to walk, and even to go to school, whilst during the summers she had been mostly bedridden.

When admitted, on July 14, 1875, she was found to be a rather undergrown child, presenting some signs of rickets in respect to the shape of the thorax. She was pale, and her face had a peculiar dirty-sallow color. The skin generally was rather dry and harsh, but free from ecchymoses, except that the front of each leg showed some ill-defined mottling.

The gums of both jaws were spongy in front of and behind the teeth. Some of the teeth were slightly loosened. The child's breath was extremely fetid. The tongue was clean.

There were no abnormal chest-signs, except that the heart's rhythm was not quite regular. The cardiac sounds were free from murmur; the pulse was one hundred and twenty in the lying posture. There were no abnormal signs in the abdomen.

The child was unable to stand, and cried when her lower limbs were moved. They were generally kept extended. She drew up her thighs when told, but very slowly. She was extremely listless when left undisturbed. The child complained most when the popliteal space was touched on either side, but no swelling could be made out there. There was no sign of any effusion into the knee- or ankle-joints. There was some deep thickening of the lower third of the right thigh, which appeared to be periosteal. This part was definitely more tender on pressure than any other part of the body.

There was no œdema of the lower limbs, nor was there any local heat of skin. Her axillary temperature on admission was 101° F. For five days it varied between 98.6° and 99.8°. Subsequently it was only on three occasions above 99°.

The child showed hysterical objection to meat and vegetables. When these were brought to her she cried, and when given to her she at first made herself sick. But a very little resolute treatment was sufficient to make her take both, and in a few days her gums underwent marked improvement and the tenderness began to subside. In six weeks' time she was sent to the Convalescent Department, her gums quite well and complexion completely altered, and she was able to stand and to walk a short distance. No other treatment than antiscorbutics was adopted. It was found, however, when the girl got about, that some thickening around the sheaths of the tendons about the ankles had taken place. This was most marked about the tendo Achillis, and there was a little pes equinus. Tenotomy was even contemplated; but within another six weeks the contraction had entirely disappeared, and also all trace of the thickening of the shaft of the femur.

Case B.—A. S. (girl), aged four years. Condition when seen: Bluish-brown staining on forehead, result evidently of an ecchymosis. Sponginess of gums, extending above the teeth in both jaws from the lateral incisors outward. Unable to stand. Great tenderness when lower limbs flexed or extended. No heat of skin; no swelling to be detected.

History: After being weaned at fourteen months, had been fed on bacon and bread, light pudding, and beef gravy. Never more than half a pint of milk in a day. Chief beverage cold tea. Had an extraordinary dislike of vegetables. Could not bear to see any vegetables on the table, or to use a spoon which had recently contained any vegetables. Would frequently go many hours refusing any food except cold tea. Had complained of pains in limbs for six months; had pointed to ankles, hips, and back. Had been unable to walk for a fortnight. A bruise-like patch appeared on forehead a fortnight ago. Gums often swollen, but worse during last fortnight.

Ordered potatoes, gravy, fresh meat, lemon juice. These were given, and the amount increased each day. In one week's time she could bear movements of the limbs and could stand leaning against the chair, and her gums were nearly well.

Case C.—James P., aged about four years. When brought, showed some rickets of wrists and ankles, and, besides this, extreme tenderness of lower limbs and inability to stand. He had a dirty-sallow complexion, spongy gums, and hysterical objection to meat and vegetables. He began to cry when a plate of meat and vegetables was procured for him, and when this food was given to him he immediately made himself vomit. Subsequently he was compelled to take it, and then kept it down. After a fortnight's perseverance with meat and vegetables, the gums were found to be natural and the tenderness of the limbs had almost vanished. The boy was then able to stand leaning against a chair.

Case D.—Mary O., aged two years and three months. A hand-fed child, the subject for the last eighteen months of severe gastric and intestinal dyspepsia. She had the most remarkable intolerance of milk, and was able to take only very small quantities of a very varied diet. She showed no naked-eye signs of rickets, and there were no evidences of tubercle. She was, so far as could be ascertained, a case of atrophy from non-assimilation of food. She had at length begun to gain a little in weight, and to keep down her food on a mixed dietary of very small quantities of the following: biscuit powder, veal jelly, Benger's food made with whey, white of egg, and raw meat. She had taken this diet for about three months, and then had been taken into the country for about a month, so that she was no longer under the writer's observation. There is reason to believe that for a few days the biscuit powder and veal jelly had been increased and the whey and raw meat diminished; but, without obvious cause or change in her circumstances, she suddenly developed sponginess of gums. When the writer saw the child a few days after the onset (at the end of the month of May), the upper gums protruded from the mouth and almost concealed the teeth, so great was the swelling. The lower gums were also swollen, but the swelling was not so extensive.

The lower limbs dropped as though they were powerless. The child screamed on the slightest pressure, but with slight examination no swelling could be made out. The front of the chest presented a remarkable appearance. There seemed to have been multiple fractures at the anterior extremities of the ribs, and the costal cartilages appeared to have sunk back away from the ribs, so that they with the sternum were on a plane posterior to their normal situation. This had taken place within the last few days without any obvious cause. The child was excessively pallid, but presented no ecchymoses. She lay on her back, and screamed with the slightest movement. She was ordered as additions to her food three dessertspoonfuls of boiled and sieved cabbage and the juice of one lemon daily. This the child took most greedily. In four days there was marked improvement, and in six days the gums were much less red and swollen and the tenderness of the limbs was so much lessened that she could be raised. The vegetables were increased and red gravy given, and the quantity of whey was also increased. In a fortnight more the child was able to sit up, and could chew malted rusk moistened with gravy. The swelling of the gums had almost vanished, and likewise the tenderness of the limbs. Her color was so much improved that there was even a touch of red in the cheeks, and she was able to assimilate her food better than had been the case for months. Six weeks afterwards, when seen again, I found, to my great surprise, that the ribs were reunited in natural position with the costal cartilages and that the shape of the anterior part of the chest was quite normal. There was no enlargement of the lower ends of the radii. She had cut two fresh teeth, and the gums were absolutely healthy. The limbs were perfectly free from any tenderness.

Case E.—Lilian W., aged two years. Shows some signs of rickets, but also some swelling of shaft of right femur, with extreme tenderness and immobility of the right lower limb. Had been suckled twelve months, subsequently fed on beef tea, bread, and some milk. Child had the greatest dislike to fruit or vegetables or meat. *The gums became swollen after the swelling of the thigh.* There was some nose-bleeding, but no ecchymosis. Vegetables and meat were given, with much difficulty. In fourteen days some improvement, and in seventeen days decided diminution of swelling and tenderness.

GROUP II.—ILLUSTRATIVE CASES IN INFANCY.

(a) *Those without Post-Mortem Verification.*

Case F.—A. B., a boy aged fifteen months. When first seen, in December, was excessively pale and sallow, lying on his back, with his left thigh kept slightly flexed and the right extended. He scarcely moves any part of his body except the head. He moans a great deal both night and day, screams if he is approached, and still more if he is touched. Both the left thigh and left leg are slightly swollen, so that the contour of the limb is different from natural, assuming in the thigh rather a cylindrical shape. Although very shiny and giving an impression of being tightly distended, the thigh and leg do not pit on pressure. There is no local heat or redness. There is no sign of fluctuation and no sign of effusion into any of the joints of the limb. The epiphyses at the knee and ankle are enlarged. The right thigh is free from swelling; the epiphyses at the right knee are enlarged. The right leg is free from general swelling, but there is distinct thickening to be felt down the shaft of the right tibia. Both radii are enlarged at the lower end, but the right more so than the left in circumference and in vertical measurement. It drops as though paralyzed, and is very tender on pressure. There is profuse head-sweating; a little thickening about the frontal region. The thorax presents definite beads. The child has cut the two lower incisors only. *There is no sponginess or ecchymosis of the gums.* The rectal temperature is 101° F. at seven P.M.

The history was the following. The only child of a fairly healthy young couple in good circumstances and living in a healthy house in town. The child was suckled six weeks only, and seemed vigorous. His mother's milk then suddenly stopped, and from that time till the period when I saw him—viz., during twelve and a half months—he had had no fresh food. At first his diet consisted of grits and Swiss milk, then of baked flour, then of Nestlé's food, then of Robb's biscuits, then of Liebig's extract, and finally of Swiss milk and saccharated lime-water. He had been considered a healthy child, although it was admitted that he had always been pale, that his stools had been unduly offensive, and that he had sweated much about the head since he was three months old. He had cut his first tooth at twelve months and his second at thirteen months. The child had been able to sit up and stand with assistance at thirteen months old. Five weeks ago he ceased to do either, and his left leg became swollen about the ankle. He became very peevish, and screamed directly he was touched. He was then taken to a well-known bonesetter, who said one of the bones of the spine was "out" and that an operation would be required to restore it to its proper position. Five days later the said operation was performed under chloroform; but, as the swelling of the left lower limb increased and the right wrist became swollen and the right hand dropped, it was determined to seek a further opinion, and then it was that the child's condition was found as above described. The view taken by the writer on the ground of post-mortems on other cases to be subsequently detailed was that the child had been for many months the subject of rickets, upon which had supervened scurvy. It was believed that there was blood effused beneath the periosteum of the left femur and tibia, and that the tenseness of this limb was due to effusion of blood into the deeper layers of the muscles and serum into the superficial layers. It seemed probable that there was also some blood-effusion around the shaft of the right tibia and also in the neighborhood of the junction of the shaft and the lower epiphysis of the right radius.

The limbs were ordered to be invested with wet compresses tightly wrung out, and these to be surrounded with dry cloths. A complete change was made in the diet. The juice of a quarter of a pound of raw meat was ordered to be given daily. A pint and a half of cow's milk was to be given in the twenty-four hours, the alternate meals having a little strained gruel or a little barley-water added. Two teaspoonfuls of orange juice were to be given daily. In three days' time the most striking change had occurred. The compresses seemed to have soothed the limbs, and he had taken the food greedily and without any indigestion. The rectal temperature had sunk to 99.4° F. The meat juice was gradually diminished, and the orange juice increased, as well as other vegetables given. After the result of the food had been conclusively established, a little cod-liver oil was given. The compresses were discontinued in a fortnight, the *general tenseness* of the left lower limb having then subsided. It was by this time easy to appreciate that there was thickening around the shaft of the left femur and left tibia, of the same kind as that felt in the right tibia at first, but greater in amount. At the end of six weeks there was still some thickening to be felt, although all tenderness had gone. Before this, also, the difference in size between the lower ends of the two radii had disappeared. After a month's time shampooing and douches were commenced, and within two months the boy would voluntarily get on his knees and stand with a little support. His face was ruddy, and his skin and muscles were becoming firm.

Case G.—Mary C., aged ten months, when sent to me was extremely anæmic, and evidently in pain on the least movement. The legs hung down in a helpless fashion, and were extremely tender. There was distinct cylindrical swelling around the shaft of each tibia. Gums were spongy, but only over and around the two lower incisors, which have been cut, and over the situation of the on-coming upper incisors, one of which is just emerging. Presented marked rickets, anterior and posterior beads on ribs, head-sweating, and enlarged epiphyses. The history given by the mother was to the effect that the child had never had the breast; that it had been fed first on Nestlé's food, then on Ridge's food, then on Savory & Moore's food, but that she had always had some diluted cow's milk. Her present symptoms had started four months ago with great tenderness and swelling of the lower limbs, which had somewhat abated lately. Six weeks ago swelling of the lower gums had first appeared. One month ago there had been blueness and puffiness of the left eye, which had gradually subsided.

The child was ordered fresh milk, with boiled sieved potato and orange juice; and the doctor who had sent her reported subsequently that the subsidence of the tenderness of the limbs and of the sponginess of the gums was most manifest, even within a couple of days.

Case H.—Fred. O., aged eight months, was sent to me by an eye-surgeon on account of proptosis of one eyeball, with great tenderness of limbs and general cachexia. Healthy at birth. No breast-milk. Fed during first three months on Ridge's food with cow's milk and water equal parts, then on Savory & Moore's food with milk and water. Liable to vomiting. Bowels not generally relaxed, but offensive. Two lower median incisors when seven months old. Mother thought the child was all right till four weeks ago, when whole body became very pale and sallow. Had been rather tender in the legs since he was quite young, but during the last week the tenderness had become excessive. The swelling of the upper eyelids had come on suddenly three weeks ago. The screaming during the last week had been almost incessant.

The child when admitted into hospital was extremely anæmic and sallow. The right upper lid was of a purplish-red color, due to deep extravasation into its substance; there was no ecchymosis of palpebral or ocular conjunctiva, but there was proptosis of the eyeball to a slight but definite amount, suggesting that there was something in the orbit pressing the eyeball downward and forward. The left upper eyelid was also a little brownish purple in color, the result of former extravasation, but there was no proptosis of the eyeball. The gums around the two lower median incisors, which are the only teeth present, are purplish and slightly raised from recent extravasation. The lower limbs were moved voluntarily a very little; they generally lay everted, with the knees slightly flexed. The epiphyses at the knees and ankles were all enlarged, and the tibiæ showed slight internal beading, but the limbs were so sensitive to the slightest touch that a proper examination of the shafts could not be made. The upper limbs were moved by the child with much more freedom, and they were obviously not so tender. All the epiphyses were enlarged. The thorax was typically rickety, with anterior and posterior beats well marked. There were no visceral signs, except that the urine was slightly albuminous and gave a definite blood-reaction to ozonic ether and guaiacum. Temperature 101.6° F.

The child was ordered one pint of milk, some mashed and sieved potato, a little gravy, the juice of one lemon, and one tablespoonful of raw meat juice, daily. In three days the change was most remarkable. The child took the fresh food quite greedily. She slept very much better. The tenderness had greatly diminished. The ecchymosis of the eyelids was lessened, and the proptosis also, to a slight extent. The ecchymosis around the lower median incisors was lessened, and no fresh ecchymosis was visible. In four days more the child sat up in bed, moved her limbs quite freely, and allowed them to be handled without crying. Her color was greatly improved, the proptosis was lessened, and the albumen and blood had vanished from the urine.

I may here state that slight albuminuria with a trace of blood was present in another of my cases, and in one communicated to me by Mr. Shoppee, also in two of Dr. Cheadle's and in one of Dr. Gee's.

(*b*) *Cases with Post-Mortem Verification.*

Case I.—Lillie S., aged ten months, was brought to me as an out-patient in October, 1881. She had never been suckled, but had been fed first on condensed milk, then on cow's milk, then on a succession of "infants' foods." At the time when she was brought she was taking Anglo-Swiss food. There had been much head-sweating since she was three months old. The bowels had been constipated until two months before, when she had a severe attack of diarrhœa. After this her legs became very tender. Three weeks ago her wrists also became very tender. The child when brought was extremely fretful: she screamed when she was approached, and still more when she was touched. The temperature was 99.8° F. Her skin was pale to the last degree. There were ecchymoses in both upper eyelids; also underneath the mucous membrane of the gums in the lower median incisor regions, and in the lower molar regions, separate ecchymoses were seen. The child had not cut any teeth, but these ecchymoses were evidently in the situations of on-coming teeth. The lower end of each radius was much enlarged, and the left hand hung prone in a condition of pseudo-paralysis. The left thigh was strongly flexed. There was deep thickening to be felt along the lower third of the shaft of the left femur. The epiphyses of the lower limbs were a little enlarged. There were rather prominent projections along junction of costal cartilages with ribs. The child lay on her back, and made no attempt to move. It was not expected that she would live, but the mother was ordered to give her raw meat juice and continue the cod-liver oil. In a week's time she was not worse, with the exception that there was now slight proptosis of the left eyeball, as though there might have been some extravasation into the orbit. Eleven days afterwards this had subsided; but the child gradually sunk three weeks after having been first seen, and about three months after the onset of her illness.

Post-mortem examination showed on both parietals a patch of subperiosteal hemorrhage about the size of a shilling, the bone beneath it being a little porous. The muscular walls of the thorax were pale yellow and watery, as though they had been bathed in serum. The periosteum of the ribs was extensively detached, thickened, vascular, and a little granular. It was separated from the rib by a considerable quantity of chocolate-colored débris, evidently broken-down blood-clots. There was no lymph or pus. The ribs were extensively bare and white. They were distinctly wasted. What had been taken during life for beads proved to be simply the ends of the costal cartilages abutting against ribs which were so extremely wasted that their anterior ends by no means came into complete apposition with the whole of the ends of the costal cartilages. There were no beads at the posterior surface. It was a wonder that the ribs had not separated from the costal cartilages or fractured beyond, they were so exceedingly brittle. There was, in fact, nothing but a shell of bone containing a little soft red medulla. On the parietal pleura of both sides there were numerous petechiæ corresponding with the ribs. There was some blood-stained serum in the left pleural cavity, but no lymph. In the middle of the left lung there were two or three very small masses of caseous tubercle and a few gray granulations on the surface. There was no tubercle elsewhere, and no disease of the other viscera. Only a partial examination of the limbs was permissible, but some blood-extravasation was found into the periosteum of the ilium and into the muscles attached to it, whilst the superficial parts were pale yellow and pulpy. There was also subperiosteal hemorrhage in the region of the junction-area of the upper epiphysis with the shaft.

The above post-mortem record need only be supplemented by a few details derived from three other cases, in two of which the examination was made by myself and in one by my friend Dr. Stephen Mackenzie. The age of each of these three cases was below two years.

The lower limbs showed, on section, yellowish serum infiltrating the upper muscular layers of the thigh and leg, the muscles pale and slightly pulpy. Deeper muscular layers contained a little disseminated blood-clot. The periosteum of the femora and tibiæ thickened, vascular, and separated from the affected shafts in great measure by sheaths of blood-clot. In all three cases, fractures through the area just above the junction of shaft with epiphysis. No callus. The two bony surfaces rough, but not splintered. The medulla of the shaft soft and red, and the trabecular structure scanty and friable.

Upper extremities.—In one of my cases, extensive extravasations of blood under the periosteum of both surfaces of the scapula. Slight deposit of new bone formed by the upraised periosteum. The bones of the upper limbs not so profoundly affected as the lower, but fracture found in one case below the upper epiphysis of the humerus.

Some evidence of antecedent rickets in all the bones. Rib changes as described in the previous case.

Visceral changes.—In addition to those before mentioned, a varying amount of interstitial hemorrhage in lungs, spleen, kidney, intestinal glands.

SUMMARY OF CONCLUSIONS.

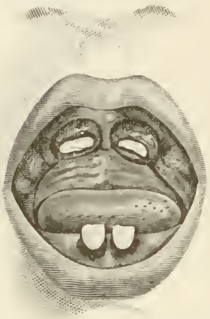
Consideration of Groups I. and II. will show (assuming that all the cases are truly scorbutic) an interesting modification of symptoms, varying to a considerable extent with the respective ages of the patients.

Taking Group I., which includes cases ranging from ten years to two years of age, we find in Case A a combination of symptoms very fairly corresponding with a chronic recurrent example of the adult type of scurvy. The sallow anæmia, the fetid spongy gums, the group of symptoms referred to the lower limbs, the syncopal (?) attacks, the extreme languor, the history of (voluntary) privation of vegetables and the striking improvement on their forcible administration, are all sufficiently characteristic to leave no doubt as to the nature of the case. It is noteworthy that the sponginess of the gums in her case comes to a maximum, and that although there was a little deep thickening detected on one femur which ultimately subsided, yet the pain and continuous distress were not excessive.

In Case B and Case C the gum-symptoms were marked, and the tenderness of the lower limbs with inability to stand was also present, but no deep swelling of the lower limbs could be made out.

In Case D the gum-symptoms were very marked, but the bone-lesions were very striking indeed. The appearances presented by the ribs, read in the light of Case H, in which there was a post-mortem examination, leave no doubt that there was fracture of ribs, with extravasation of blood near the fractures, and that on the recovery of the child these fractures were completely repaired.

FIG. 1.



SPONGY GUMS FROM A CHILD AGED FOURTEEN MONTHS, THE SUBJECT OF INFANTILE SCURVY.—The picture shows the extremely slight character of the lesion and its limitation to the tissues around the teeth and over the on-coming teeth. In the upper gum there is sponginess only around the upper median incisors. At the extremities of the upper gum are to be seen slight ecchymoses in the gum, without sponginess, and in the lower gum, around the median incisors, there are also ecchymoses without sponginess.

Case D is approaching the *infantile* group, in which the bone-symptoms come into greater relief.

Case E, though slight, is of importance because the lower-limb manifestations *appear in order of time before the swelling of the gums.*

In Group II. the gum-symptoms begin to recede into the background, whilst the bone-symptoms, including those of the lower limbs, but also *other bones of the body*, become the dominant characters. The gum-symptoms are seen to have a very definite relation to the number of teeth which have been cut. There may be swelling and sponginess around the teeth, but on the portions of gum where no teeth have been actually cut there is generally no sponginess. In some cases there are ecchymoses in the sites of on-coming teeth. This is illustrated in Case I. and in the accompanying picture of another case under the care of the writer (see Fig. 1). It also obtained in one of Dr. Gee's cases. These ecchymoses may be exceedingly transient, and are very liable to be overlooked.

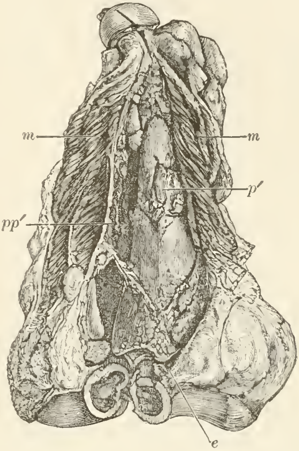
As sponginess of the gums is generally regarded as a crucial test of scurvy, it is of the first importance to point out that in ordinary adult cases of scurvy *no sponginess* appears in portions of the gums from which teeth have been extracted, and, further, that an edentulous adult when he suffers from scurvy does not get sponginess of the gums at all. (See Immermann and others.) In some of the cases in this group there was neither sponginess nor ecchymosis of the gums,—*e.g.*, Case F,—but it is maintained that the other symptoms were so marked, and the almost instantaneous amelioration by the use of fresh vegetables was so striking, that they are also examples of scurvy.

With respect to the bone-symptoms the infantile group shows very remarkable changes. In the light of the four post-mortem records, it may be briefly stated that the primary scorbutic bone-change appears to be that there is blood-extravasation between active growing periosteum and the subjacent bone.

Such extravasation is seen best in the lower limbs (see Fig. 2). With respect to the thigh, for example, there may be a more or less complete sheath of blood-clot between the upraised periosteum and the greater part of the shaft. The shaft then, being partly unsupported and also deprived to some extent of its nutrient supply, may undergo a degree of atrophy, and may also with the minimum of violence undergo fracture. The common situation of such fractures is just above the junction-area of shaft with epiphysis, but they may also occur in the middle of the shaft.

The above conditions may also be found in the tibia, and, much less frequently, in the bones of the upper extremity. They may also be found in the ribs, giving rise to the remarkable features of the front of the chest before described.

Fig. 2.



Femur and Surrounding Tissues, showing Results of Scurvy in a Child who was also the Subject of Rickets. From one of Dr. Barlow's cases.—Child aged twenty-one months at death. Duration of symptoms of scurvy, two months. *m*, muscles, the upper layers infiltrated with serum, the deeper layers containing blood-clot; *pp′*, thickened vascular periosteum separated (except at the upper end) from the shaft by masses of blood-clot; *p′*, blood-clot investing the shaft; *e*, lower epiphysis, with a very small portion of the shaft separated by fracture from the main portion of the shaft. No callus. The drawing is copied, by permission, from Mr. Erichsen's "System of Surgery."

The way in which these fractures repair under simple rest and antiscorbutic diet vigorously pushed is very remarkable (see Case D).

It is also noteworthy that the retention of the osteogenic power of the upraised periosteum in some of these cases will explain the bony sheath which can sometimes be detected in the long bones for a time after all active symptoms have subsided. This is illustrated in Case F and in the account given in the paragraph on post-mortem appearances of the scapula in one case.

The writer believes that he has evidence that the proptosis which is recorded in Case H and Case I, and of which he has seen other examples, depended also on a bone-lesion,—viz., extravasation of blood between the orbital plate of the frontal and its subjacent periosteum, this extravasation tending to push down the eyeball.

In connection with the various hemorrhagic extravasations under periosteum, it becomes easy to explain the extreme tenderness and distress occurring in the infantile group, and also in some members of the childhood group, as compared with what obtains in adults. Post-mortem examinations indeed have proved, in adults suffering from scurvy (especially in young adults), that hemorrhagic extravasations may occur between bone and periosteum (see one of Lind's cases, and Budd's case, also statement of Vidal on subperiosteal hemorrhage in scurvy in the last epidemic at Paris, during the siege of 1870. With regard to fractures near epiphyses in young adults in scurvy, see observations of Poupart in a Paris epidemic in the seventeenth century, and with regard to the fractures of the ribs in scurvy, see some of the Russian reports). But these occurrences are probably far from constant. The brawny indurations in adults seem to depend on effusions into muscles and cellular tissue, and they are attended with less pain than if there were a tight extravasation under the periosteum.

The remarkable responsiveness of the actively-growing bone-tissues in infancy to any altered blood-state seems to explain in some degree why the limbs should be so much more profoundly altered in infantile than in adult scurvy. The various internal hemorrhages (into pleura, lung, spleen, glands) referred to in the post-mortem accounts are quite in harmony with the statements given as to adult scurvy.

With regard to the circumstances under which the affection has arisen, the examples given in the childhood group are quite similar to those of many cases of adult land scurvy; and it is interesting to note, by the way, that some of the children affected appear to have had a curious hysterical dislike to antiscorbutic food. But with regard to the infant group there may at first sight appear some difficulty in accepting the parallelism. Let it be noted, in the first place, that there is no evidence that any child has developed this group of symptoms whilst being suckled at the breast. Further, the most striking cases are those in which infants have been brought up for several months on artificial foods prepared with water, and without any fresh aliment. In a number of cases it is true that the disease has developed when children were taking artificial foods prepared with a varying amount of cow's milk. Now, cow's milk has undoubtedly antiscorbutic power if given fresh and in sufficient quantity; but there comes into question, with regard to infant feeding, the difficulty as to how much the milk has been diluted. It is also noteworthy that the group of symptoms in question are very apt to supervene upon obvious or latent rickets. This seems to play the same part in the infant cases which the debilitating influences of syphilis, malaria, dysentery, damp and cold, etc., play in adult scurvy. The true cause is a deprivation of fresh food; but the presence of rickets, like other states of lowered nutrition, predisposes to scurvy. The most striking parallelism is to be found in the responsiveness to fresh food which characterizes alike the infantile and the adult group. Juice of fruits and strained vegetables are taken with avidity, and the most marked alteration is produced by these alone. Fresh cow's milk and raw-meat juice are also frequently assimilated in a very striking manner. Whilst the evidences of rickets remain and take months to recover, the tendency to ecchymosis in the gums and elsewhere is suddenly arrested, and the pain in connection with the limbs soon diminishes, although the thickening of the shafts takes time to subside. If the cachexia be very profound the child may succumb, but the cases are rarely fatal except from intercurrent malady. For a more complete discussion of this subject the writer refers to his paper published elsewhere (for reference *vide postea*). Without maintaining that rickets is never initiated in an acute fashion, the writer has attempted to prove that many of the cases described by the German writers under the title of "acute rickets" are, strictly speaking, a "combination of scurvy and rickets, the scurvy being an *essential* and the rickets a *variable* element." The writer is of opinion that in no disease (not even syphilis) is the therapeutic test of more value in diagnosis than in the present group.

(1) Predominance of lower-limb affection: (*a*) immobility, going on to pseudo-paralysis; (*b*) excessive tenderness; (*c*) general swelling of lower limbs; (*d*) skin shiny and tense, but seldom pitting, and not characterized by undue local heat; (*e*) on subsidence, revealing a deep thickening of the shafts; (*f*) liability to fractures near the epiphyses. (2) Swellings of gums, varying from definite sponginess down to a vanishing-point of minute transient ecchymosis. These constitute the chief diagnostic differentia between infantile scurvy and rickets properly so called. But to them must be added, as the most important diagnostic of all, (3) definite and rapid amelioration by antiscorbutic regimen.

SUMMARY OF TREATMENT.

The general lines of treatment, as indicated in the foregoing cases, consist in the administration of fresh milk instead of condensed milk and the artificial infant foods. The milk (cow's, ass's, or goat's, as may be found best) should be given with as little dilution as possible. In these cases milk is often assimilated without any dilution at all. To the milk should be added thoroughly-boiled, carefully-sieved potato. Orange juice, beginning with the juice of one orange daily, may be given even to the youngest infant attacked with this disease. Fresh raw-meat juice may be given to the amount of one or two teaspoonfuls a day at the beginning of the treatment.

To children over twelve months old, boiled sieved green vegetable may be given advantageously, with milk or cream; and the writer has given it even to younger children than this, with much benefit.

Locally, fixation of lower limbs is important, and for this purpose sand-bags are often adequate. Occasionally wrung-out wet compresses surrounded by dry cloths are useful. Exposure to fresh air is very valuable, if the horizontal position can be properly maintained. In all cases the greatest possible ventilation ought to be secured.

BIBLIOGRAPHY.

There is no space within the limits of this article to give any account of bibliography, but the writer must point out that among English physicians the great merit of having first shown on clinical grounds the true affinities of this form of infantile cachexia undoubtedly belongs to Dr. W. B. Cheadle. (*Lancet*, November, 1878, Three Cases of Scurvy supervening on Rickets in Young Children. *Lancet*, July, 1882, Osteal or Periosteal Cachexia and Scurvy.) Among Continental reports Dr. Ingerslev's contribution, Case of Infantile Scurvy (child aged fifteen months), given in Virchow's *Jahresbericht*, 1873, p. 697, though extremely brief, is very important.

The essential part of the writer's personal contribution to the subject consists in the account of the anatomical nature of the disease as determined by post-mortem examination, which anatomical conditions explain in great measure the curious grouping of symptoms belonging to the lower limbs (*Medico-Chirurgical Transactions of London*, vol. lxvi., 1883, On Cases described as "Acute Rickets," which are probably a combination of scurvy and rickets, the scurvy being an essential and the rickets a variable element). The anatomical conditions described by the writer have been verified in other cases by several observers,—Stephen Mackenzie, Page, Colcott Fox, and quite lately by Rehn.

CRETINISM.

By JUDSON S. BURY, M.D., M.R.C.P.

THE origin of the word cretin is uncertain: some derive it from *chrétien*, because of the happy disposition; others from *crétira*, "stupid," or "silly;" others from *creta*, because of the chalky complexion, or in allusion to the calcareous nature of the soil; while Esquirol, believing an alluvial region to be one of the chief causes of the malady, referred the derivation to *crétine*.

Definition.—Cretinism is a chronic disease, for the most part endemic, in which, associated with a varying degree of mental and moral torpor, there is a characteristic physiognomy and a peculiar malformation of the head and body, dependent to a considerable extent on premature union and arrested growth of certain bones, and having an intimate though obscure relation to disease or absence of the thyroid gland.

History.—The history of cretinism prior to the sixteenth century is enveloped in obscurity, and it is not till towards the end of the eighteenth century, when Malacarne's important work appeared, that we begin to find scientific accounts of the disease; but since that date the literature of the subject is very extensive.

Geographical Distribution.—Endemic cretinism is met with in almost every part of the habitable globe. On European soil the headquarters of cretinism and also of goitre are the Alps, the Pyrenees, the Vosges, and the Jura. In Italy its centres are the valleys of the Alpine chain traversing Piedmont, Lombardy, and Venetia, the district of Aosta at the foot of Mont Blanc, and the northern slopes of the Apennines. It is found in France mainly in the departments of Hautes-Alpes and Savoie, with a percentage of 1.6 to 2.2. It is also prevalent in the Hautes-Pyrénées. Although goitre and cretinism are usually found in the same districts, it is remarkable that in the hilly country of the Aisne, where goitre abounds, the cases of cretinism are very few. In Switzerland the disease is worst in Uri and in the Valais, having a percentage of .6 to .9. It is also common in Bern and in several other places.

In Spain goitre and cretinism are widely distributed, and are especially common among the valleys on the southern slope of the Pyrenees. In Austria both goitre and cretinism have their chief seats on the banks of the

Danube and the Traun and in the valley of the Leitha. In Hungary, although goitre is endemic in many places, cretinism occurs chiefly sporadically. In Germany the two diseases have a wider diffusion in Würtemberg than in Bavaria; they occur in the circle of the Black Forest, but are absent at its highest points. The diseases are comparatively rare in Central and Northern Germany, the plain of North Germany and of the Netherlands being quite free from endemics. Cretinism is also rare in Belgium. While goitre is pretty common in England, cretinism is rare; sporadic cases are met with, but the endemic centre formerly existing at Chiselborough in Somerset is now extinct. In Scotland cretinism and goitre are found on the east side of Arran and on the east coast of Fife. In Sweden a few centres of goitre occur, but Norway and Denmark are free from endemic goitre and cretinism.

In Russia both diseases are met with in the valley of the Ojat, goitre also in a few other places. In Siberia they are commoner, especially in the government of Irkutsk.

On the continent of Asia the centres of cretinism and goitre are the northern and southern slopes of the Himalaya Mountains; they are also endemic in the northern provinces of China, and in the mountainous parts of Burmah and Cochin China.

With regard to Africa there is no exact information; cretinism is said to exist in Madagascar. In North America cretinism is not common except at a few points,—namely, in the valleys of Vermont, in Massachusetts, and in California.

In South America it prevails in the course of the Magdalena River, but is much more limited in area than goitre.

Influence of Locality.—Although widely spread over the globe, and in certain parts covering large tracts of country, the seat of endemic goitre or cretinism is always narrowly limited to a few spots, outside which even in the immediate neighborhood there is complete immunity: thus, a particular village may suffer, while an adjoining locality is exempt. It is asserted that healthy parents coming to live in affected spots are likely to propagate cretinous children, and that if goitrous or healthy parents remove from regions where goitre and cretinism prevail they will beget healthy children.

Influence of Climate, etc.—Both diseases occur in all latitudes, from the equator (as in South America) to the Arctic zone (as in the Hudson Bay Territory). They appear to be independent of climate, season, or weather. Some authorities have regarded excess of moisture of the atmosphere as an essential condition; but it must be remembered that the diseases occur in places where the atmosphere is absolutely dry, as in some parts of Brazil.

Altitude, etc.—Goitre and cretinism are chiefly endemic in mountainous regions, especially in high mountain-ranges, such as the Himalaya, the Alps, and the Cordillera, while they are rare at low levels and are never found close to the coast. Nor are they so prevalent as was formerly thought in damp, sunless, deeply-cleft valleys; and the presence of cretinism at Langenargen on the Lake of Constance is a striking example of the disease developing on an open plain.

Relation to Soil.—The question of the geological character of the soil has been much discussed, but we want more accurate information as to the condition of the soil, and not only of the upper layers but also of the lower ones. Cretinism and goitre are found on every geological formation, but favor the older more than the newer formations, and especially the sedimentary ones composed of the detritus of older rocks, as, for example, the plains of Lombardy and the Rhine. But it is noteworthy that in a district where the geological formation is the same in every part, cretinism or goitre may be prevalent in some points and completely absent in others, proving that their occurrence is not dependent solely on geological formation. The importance of *wet soil* as a causal factor has often been insisted upon, and without doubt the decrease of goitre and cretinism in the valleys of Savoy and other places is largely owing to improved drainage. At the same time, both maladies are met with fully developed in dry and sunny situations, as the district of Aigle in the Rhone valley; while the marshes and moors of low countries are sometimes quite exempt.

Connection with Limestone and Dolomite Soil.—One of the oldest and most popular doctrines with regard to the origin of goitre and cretinism is their dependence on drinking-water rich in lime salts. It is incontestable that water rich in lime and magnesia is very common in the endemic regions, for the careful investigations of Grange and others have proved that the diseases in question occur, though not exclusively, yet for the most part, upon limestone and dolomite soil, the latter, or magnesian-limestone rock, being the more important of the two. But, on the other hand, numerous observations show that, although the connection is very close, the pathogenesis of goitre and cretinism does not depend on these factors. Thus, there are many places in which these diseases prevail where the drinking-water is almost free from mineral constituents, as, for example, in the Salzburg Alps; and, indeed, it is stated that goitre in Switzerland is much more frequent in places where the water is poor in lime than in those where the quantity of lime is great, also that in districts in the Punjab subject to goitre and cretinism the drinking-water is almost pure. Again, in New Zealand, where there are large masses of magnesian limestone, goitre is quite unknown.

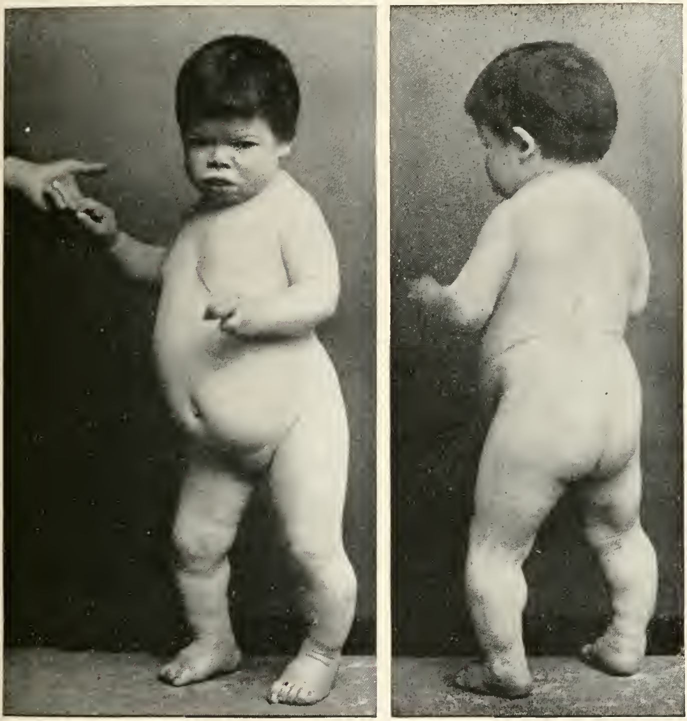
From the above review of the conditions under which cretinism is stated to occur, we see that the disease may be met with in any climate, in any locality, and that it appears to bear no fixed relation to soil or to altitude. In future investigations into the causation of the disease, the question of heredity should be reconsidered, *and especially should it be ascertained to what extent close interbreeding takes place in the affected districts.*

Symptomatology.—*Endemic Cretinism.*—In some cases many of the characteristic features of the disease are present at birth, but in the majority are not recognizable till the sixth month, when the following symptoms may be observed. The child is weak; its body is fat and puffed out; the skin is generally brown or of an ashy tint; the head is large, and the fontanel, and sutures are widely open; the eyes are languid and expressionless and generally half closed; the appetite is voracious; the child is sleepy and apathetic, and appears indifferent to light and sound; the mouth is large, the lips are thick and swollen, and the nose is short and broad; the child rarely cries, and the cry has a peculiar hollow sound; the belly is tumid; the limbs are usually small and weak; the neck is thick and short, and sometimes presents an enlarged thyroid. Growth is slow; the teeth are cut late, and their development is tardy and irregular; they easily blacken, decay, and fall out, often never to be replaced; dentition is usually accompanied by abundant salivation, and often by violent convulsions. Later many of the above symptoms become more pronounced, and the child rarely walks till the sixth or seventh year. About this time, or a little later, the young cretin, if not deaf from birth, begins to articulate certain sounds in a hoarse, shrill voice. The seventh year is considered by Dr. Morel to be the critical age for cretinism, for it is most exceptional for the disease to develop after that period. Puberty is late or does not appear at all; occasionally its advent has a favorable influence on the milder forms of cretinism.

We will now consider the more important features of the fully-developed disease. In regard to *stature*, cretins rarely exceed four feet eleven inches, and many are only about three feet in height; some are stated to have reached a normal standard, and a few to have exceeded this, in exceptional cases even attaining a height of six feet, but the true nature of the last-mentioned is open to question, the term cretin being often too loosely applied to all persons of feeble intellect.

Besides being short, cretins are usually deformed, and in consequence of defective development often show a disproportion in size between all or certain parts of the body. The trunk is relatively longer than natural, the thorax generally large and flattened, the breasts in the female pendulous and flaccid, and the belly voluminous. The limbs are often crooked, and the ends of the bones enlarged; the lower limbs are short and thick, the feet flat, and the gait awkward,—called, in the German parts of Switzerland, the "Bärengang;" the hands are large and spade-like, and the fingers short, especially the thumb, while the nails are usually large, flat, and brittle.

The *head* is big, if not absolutely, at least relatively to the rest of the body, and is held erect with difficulty. In a large number of cases it is brachycephalic,—that is, contracted from before back and expanded at the sides,—and frequently measures more from ear to ear than from the root of the nose to the occipital spine; the top is flattened, and the occipital region often especially so. In advanced cretinism there is sometimes a transverse sulcus just above the level of the eyebrows. The hair, commonly a dirty chestnut color, and rough, coarse, and bristly, extends low down on the forehead. Cretins seldom become bald, and the hair rarely white. In other



Front view. Back view.

"Sarah" (under Dr. Shuttleworth, Lancaster), aged Twenty-one Years when Photograph was taken.—Seen by the writer through the kindness of Dr. Shuttleworth. Features characteristic. No goitre; fulness of neck, but no distinct fatty tumors. Frontal suture and pit over anterior fontanel not completely closed. Loose skin. Can speak a word or two, but very slowly. Shy, affectionate, and somewhat jealous. All tendon-reactions increased. Height, nearly three feet. Weight, forty-nine pounds. Slight menstruation twice in 1888. No pubic hair.

parts of the body, as the chin, the axillæ, and the genitals, there is usually but a scanty development of hair.

The *face*, somewhat resembling the Mongolian type, is square and large, especially in the upper third, and the expression is stupid and monotonous. The nose is short, depressed at its root, and spreads out enormously towards the alæ, the nostrils being widely open. The eyes are widely separated, and, in addition to their bleared look, are usually affected with strabismus; the lids are often swollen and scarcely open; the eyelashes are short and scanty, rarely bushy and tangled; the eyebrows are also thin and irregular. The zygomatic arch is broad, and the upper jaw prominent, the inferior maxilla small, retreating, and its angle very obtuse. The tongue, large, swollen, and sticky, usually protrudes from the large, widely-open mouth; the lips are thick and flabby, the lower one hanging down and frequently dripping with saliva. The skin of the face, at first a dull livid white, becomes a yellowish brown, and is coarse, rough, and wrinkled.

The pinnæ of the ears are large, deformed, and unduly separated from the head.

The *neck*, besides being short, thick, and fat, presents in a large proportion of cases a goitrous tumor. The relation between the condition of the thyroid gland and cretinism will be discussed in a subsequent section, but it may be here remarked that goitre is absent in one-third of the cases of cretinism, that its size and consistence vary within wide limits, that its size and the frequency with which it occurs in association with cretinism usually bear an inverse proportion to the intensity of the latter disease, and that in some cases all traces of a thyroid gland are completely absent.

The *skin* is of a tawny yellowish color, rough, thickened, and wrinkled, and looks as if too large for the body. There is also a great increase of the subcutaneous fat, and the muscles feel soft and flabby. The slow, waddling gait has been already mentioned; other voluntary movements are equally sluggish and undecided; there is an inability to stand long, owing to general muscular weakness. All the vital functions are languidly performed. The pulse is often very slow, and the temperature lower than normal. The digestive functions, in spite of the voracious appetite and the imperfect mastication and salivation of the food, are not perceptibly disturbed. Respiration is slow and often embarrassed, sometimes in consequence of pressure on the trachea from an enlarged thyroid, sometimes from an accumulation of mucus in the bronchi which the cretin has not the sense to expectorate.

As to the *secretions*, the saliva is often viscid; the urine, commonly turbid and offensive, quickly undergoes ammoniacal decomposition, and is said to be poor in solids.

Menstruation, always late, may not be established till the age of twenty or twenty-five, and is usually irregular and scanty. When a cretin woman is able to give birth to a living child, she has rarely any milk to suckle it with.

With regard to the *special senses*, the eyesight is generally good, but hearing, taste, and smell are usually blunted. Scarcely a third of these unfortunates enjoy perfect hearing; the auditory foramen is often very large and blocked with wax, and in some cases completely obliterated. The cutaneous sensibility is also blunted, and the sexual function diminished or annulled. Cretins are heavy sleepers, difficult to wake, and when roused from sleep look stunned, astonished, and somewhat resemble an epileptic after a fit.

Their *mental deficiency* varies from mere stolidity to complete fatuity, and authors have divided cretins into three classes, according to the degree of their mental powers. In the first class the subjects, called *simple cretins*, manifest only vegetative functions, and are entirely destitute of reproductive and intellectual faculties, including the power of speech. The second class, called *semi-cretins*, possess the power of reproduction and some rudiments of language, but their intellectual efforts are limited to bodily wants. The third class, the *cretinous*, as they are called, have more intellectual power than the "semi-cretins," and are able in some degree to learn a trade or to do different kinds of work. The only peculiarity—and this is not constant—which distinguishes the mental condition of cretinism from that of other forms of idiocy, is one specially alluded to by Maffei,—namely, "the total suspension of almost every mental act during several hours, and that periodically several times in the day. During these attacks the cretins remain with their eyes open and fixed upon the sky or some object, without moving the eyelids, the mouth open, almost without breathing and without giving any sign of life. To see this immobility, this passivity of physiognomy, one would say that the soul had entirely left the body; in fact, a similar absence of all emotion in the countenance gives room to think that there is neither consciousness nor life within them." Such a condition reminds one of "le petit mal" in epilepsy, and it is noteworthy that cretins are very liable to suffer from eclampsia. There is nothing special with regard to other complications: hernia is common with cretins; phthisis is rare; and rickets is stated to be frequently associated with the disease.

Sporadic Cretinism.—Isolated examples of cretinism are met with in various parts of England which do not appear to bear any relation to locality. The essential features of such sporadic cases are identical with those already described as characteristic of endemic cretinism. There is the same dwarfing of the body, with disproportion and want of symmetry between its different parts. There is the same type of head and face,—the former large and broad, the latter with high cheek-bones, and eyes wide apart and set, as it were, in the ends of a transverse gutter, the middle of which takes the place of the bridge of the nose, the end of the nose broad and upturned, the mouth large and gaping, the tongue showing at its verge, or protruding and swollen, the lips thick. The hands and fingers are broad and short, and the limbs stunted; in severe fœtal types, which probably never survive birth, the upper limbs (as in one of Thomas Barlow's cases)

Fig. 1.

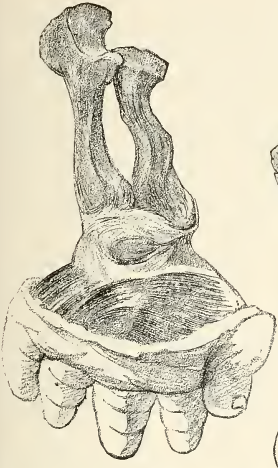


Fig. 3.

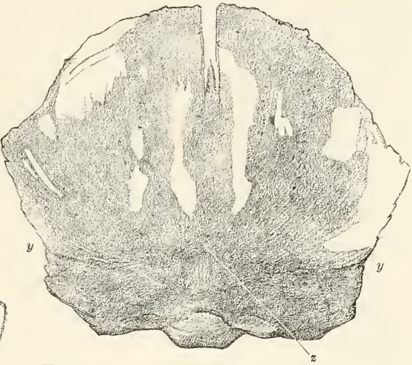


Fig. 2.

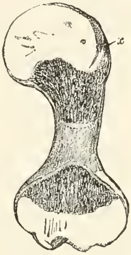


Fig. 4.

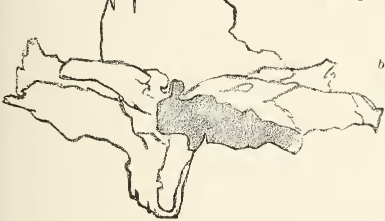
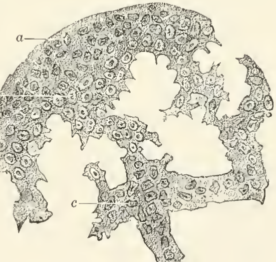


Fig. 5.



Illustrating Dr. Barlow's Case of so-called Fœtal Rickets (? Cretinism).

Fig. 1.—Forearm and hand showing radius with sigmoid curve and short stunted fingers.

Fig. 2.—Section of humerus with relatively large epiphyses and short compact shaft; line of junction quite sharp and defined, and rudimentary bony sheath forming a slight cup round the epiphysis.

Fig. 3.—Supra-occipital. *z*, internal occipital protuberance; *y*, *y*, are at the extremities of the lines which correspond with the junction of the lower part (which is developed in cartilage) with the upper part (which is developed in membrane). Thus the cartilage-formed portion is stunted as compared with the membrane-formed portion.

Fig. 4.—Vertical antero-posterior section through basis cranii, showing premature synostosis between basi-occipital and basi-sphenoid.

Fig. 5.—Section of junction of epiphysis with shaft of femur. *a*, cartilage-cells above line of ossification; *b*, cartilage-cells at line of ossification, the cells much enlarged and spherical in shape, but showing very imperfect row formation; *c*, bone trabeculæ. (Oc. 2, ob. 8. Tube in Hartnack.)

(By permission of the Pathological Society.)

may not extend further than the umbilicus, and the lower limbs measure only four inches in length; the long bones are also curved and their epiphyses frequently enlarged. The skin, as in the endemic class, is rough, wrinkled, often thrown into transverse folds, and feels loose and easily separable; this is well shown by pinching up the scalp. The thyroid gland is usually absent; sometimes it is quite normal, sometimes it is slightly swollen, but a large goitre is rarely present. Frequently soft, movable, lobulated fatty tumors occupy the angles between the sterno-mastoids and the clavicles; in some cases such deposits of fat are found in other situations, as at the nape of the neck behind the mastoids; in others the subcutaneous fat is not heaped up into separate swellings, but is generally increased throughout the body. These swellings have been observed to dwindle and disappear during exhausting illness. Fagge regarded them as a sign distinguishing sporadic from endemic cretins. McClelland, however, speaking of the latter group, says that sometimes there is a fulness of the base of the neck on one or both sides "above the clavicles," and it is possible that the swellings may have escaped the observation of other writers; one must remember, too, that such fatty tumors in the posterior triangles are occasionally present in health.

As regards the muscles, it is noteworthy that in some cases they feel enlarged and hard.

The circulation is feeble, the extremities cold, and the fingers and toes of purplish hue. A curious intermittent flushing of the cheeks and nose is sometimes seen, even when the patient is quite at rest: this was marked in "Sarah," whose photograph is appended. The temperature is low, 95° or 96° F., with a daily variation less than normal.

With regard to the nervous system but few observations are recorded in literature. Sensation appears to be retarded, but this may be accounted for by the hebetude of the patient. Slowness of movement is a characteristic feature, but actual paralysis does not occur. The gait is awkward and waddling. In three cases observed by the present writer, the knee-jerks were increased, in "Sarah" markedly so, and the tendon-reactions at the elbows and wrists were also greatly exaggerated; in this girl, too, the feet were unduly arched and the first phalanges of the toes hyperextended.

The spine is often curved; in the cases seen by the writer, the lower cervical and upper dorsal vertebræ formed a rounded prominence behind, while the lumbar region presented a deep concavity, as if dragged forward by the enlarged and protuberant abdomen.

The intelligence of sporadic cretins is very imperfect, though varying much in different cases. The slowness in apprehension is amusingly demonstrated by watching the gradual onset, slow development, and imperceptible subsidence of a smile. Many cretins are deaf and dumb, and exhibit an extreme degree of idiocy. The disposition is usually mild and inoffensive, and often affectionate; sometimes sporadic cretins are shy, jealous, and irritable. They are said not to be so unsociable and repugnant to one another as the endemic class; but such differences may be accounted for by the varying amount of care bestowed on these unfortunates.

Morbid Anatomy and Pathology.—Post-mortem reports on cases of endemic cretinism are lamentably few and imperfect. With the exception of Virchow's important observations on the condition of the skull in a cretinous infant,—which have been confirmed, as we shall presently see, in many cases of sporadic cretinism occurring in England,—we have scarcely any definite information. We do not know of any accurate record as to the condition of the *thyroid gland.* Investigators have contented themselves with reporting the existence or non-existence of a goitre during life, but apparently have not deemed it necessary to examine its structure after death. Indeed, literature abounds in discussions of the geographical and clinical associations of goitre and cretinism, but gives no post-mortem facts with regard to the presence or absence of the thyroid gland, nor does it tell us whether there is any proportion, direct or inverse, between variations in the amount of healthy thyroid gland tissues and different degrees of cretinism.

Descriptions of the brain are also far from satisfactory, and good microscopical observations still a desideratum.

With regard to the skull the most remarkable change is that first described by Virchow,—namely, a premature ossification of the spheno-basilar bone, or "os tribasilare." This in fœtal life consists of three parts, —the pre-sphenoidal, the post-sphenoidal, and the basilar process of the occipital bone,—which at this time are separated by disks of cartilage; the two segments of the sphenoid begin to unite before birth, and at birth some osseous union is present, but traces of cartilage may be found up to the thirteenth year.

The sphenoid and the basi-occipital should remain *separate* until at least the fifteenth year. Now, the fœtal skull examined by Virchow presented complete coalescence of these three bones, a continuous layer of diploë passing from one to the other and no trace of the original separation being visible. The consequences of such early synostosis are, that the base of the skull ceases to grow in an antero-posterior direction; that there is a remarkable deformity of the internal base, of which the most constant features are a narrowing of the sella turcica, and an abnormally wide angle between the clivus and the parts in front of the posterior clinoid processes; that this arrest of development hinders the growth of the base of the brain, and leads to those extensive changes in the configuration of the skull and osseous framework of the face which produce the remarkable physiognomy during life. The contraction of the skull-base is largely compensated for by expansion of the cranial vault and by delay in the closure of the sutures, the latter, together with the anterior fontanel, sometimes being found open even in adult life. The sinking at the root of the nose is probably the result of imperfect forward growth of the vomer; and a dilatation of the cavity of the body of the sphenoid bone will probably cause some atrophy of the ethmoid. Sometimes the clivus is steeper than natural, sometimes it is well-nigh horizontal, and the central furrow for the medulla may be wanting, the degree of inclination of the clivus depending to some extent on the age of the cretin, for in the healthy fœtus it is nearly vertical. (The horizontal position of the basilar process in endemic cretins is an old observation, having been described by Ackermann in 1790, Fodéré in 1792, and others.) Further, the cranial bones are often thicker than normal, the carotid and other foramina diminished in size and the occipital fossæ very shallow. The premature ossification of the basis cranii is, however, stated not to be constant, and in the skull of a female cretin aged twenty-eight years Lombroso found that the basilar process and the condyles of the occipital bone were completely absent, their place being taken by two plates of bone like the inferior articular processes of the atlas; thus, the first vertebra bounded the occipital foramen, the latter descending in a vertical direction. The dura mater is usually thickened and firmly adherent to the bone. No special or constant characters are recorded of the *nervous centres.* The following have been noted in different cases: undue flattening and simplicity of convolutions; increase of fluid at the base and in the ventricles; diminution or increase of color and consistence of brain-structure; the fissure of Sylvius ill defined and shallow; diminution or increase in size of the corpus callosum and of the basal ganglia; the cerebellum often small, asymmetrical, and its lamellæ much reduced in number,—in one case three hundred instead of six hundred; the medulla oblongata and spinal cord small; irregularities in the origins of the cerebral spinal nerves.

The subcutaneous tissue and the pale flabby muscles are usually infiltrated with serosity. The larynx, especially when compressed by a thyrocele, is diminished in capacity, and the vocal cords are often small and œdematous. The peritoneal cavity often contains a little fluid, and the mesenteric glands are engorged. The mammæ and the organs of generation are frequently rudimentary or atrophied.

Sporadic Cretinism.—Curling, in 1850, was the first to describe the morbid anatomy. In one case, aged ten years, the swellings in the neck were found to dip down behind the clavicles and to fill the axillæ; they were composed simply of fat, which was not encapsulated. In his second case, aged six months, the fatty neck-swellings were also found to be free from any investing envelope. In both cases the thyroid gland was absent. Of late years further important post-mortem observations have been recorded by Fagge, Beach, Barlow, Bowlby, and others, of which the following is a brief summary.

The skin is rough, wrinkled, and often thrown into transverse folds, sometimes of a semi-translucent and œdematous appearance. The subcutaneous fat is sometimes heaped up in masses, as in the neck-swellings; sometimes there is a general increase, the neck-swelling being absent. The freedom from investing envelope accounts for the occasional absorption of these swellings. In some cases the thyroid gland is present and quite natural, but in most cases no trace of it can be discovered.

The *skull* in many fœtal and in a few adult cretins presents the basal changes already described, but it should be remembered that they are not constant, and that the cartilaginous base may be quite normal even in fœtuses which present all the other important characters pertaining to sporadic cretinism. When the change of the base is present, it is remarkably limited to the parts formed in cartilage; the portion of the occipital bone below the occipital spine is stunted in proportion to the part above, while the latter and the other tabular bones which are formed in membrane show normal or excessive development. In addition to the premature ankylosis of the basi-occipital, the basi-sphenoid and the presphenoid bones, the clivus in the fœtus is commonly steeper than natural, and the foramen magnum usually narrowed, and funnel-shaped, cordate, or elliptical in outline. In some cases the membrane-formed bones are thin and deficient, in others all the skull-bones are thick and porous. In an adult cretin examined by Fagge there was elevation of the foramen and the basilar process, the margin of the foramen was surrounded by an elevated rim, and the cerebellar fossæ of the occipital bone were exceedingly shallow. The clivus was more horizontal than natural, the posterior clinoid processes being, however, at a much higher level than the anterior, and nearer to them than usual,—the sella turcica being greatly dwarfed in the antero-posterior direction.

The *brain* in this case was natural in appearance, and the cerebellum was not so small as might have been expected from the shallowness of the occipital fossæ. In other cases there has been an excess of fluid in the ventricles, and turbid serum in the subarachnoid tissue. In a fœtal cretin described by Thomas Barlow the brain presented remarkable features, apparently the result of the basal malformation of the skull. The crura, pons, and medulla were more vertical than normal, and the pons was laterally compressed; the cerebellum was more covered by the cerebrum than in a healthy fœtal brain; it was pushed forward and had grown more in an upward direction than is natural. There were also remarkable abnormal fissures of the temporo-sphenoidal lobes, opposite to the anterior parts of the sides of the cerebellum, and probably in relation to its upward thrust.

The *spine*, frequently abnormally curved, is otherwise usually natural; occasionally an irregular formation of bone and incomplete ankylosis of the laminæ of the arches of vertebræ have been observed.

The *clavicle*, a membrane-formed bone, is of normal length. All the other *long bones* are shortened, and in some fœtal cases are remarkably dwarfed: thus, the femur may measure one inch in length, and all the other limb-bones from one-third to three-fourths of an inch. The epiphyses are much enlarged in proportion to the shaft, and may constitute three-fourths of the whole length of the bone. The bones are usually thick and firmly ossified, and they present curvatures which are commonly exaggerations of the natural ones. *Section* of a long bone, as the humerus, shows that the bone is hard and dense and free from any evidence of fracture; that there is an invasion of fibrous tissue from the periosteum in between the epiphysis and shaft; that around the base of the epiphysis there is sometimes a sheath-like prolongation, which may even be ossified, forming a distinct cup around the epiphysis. The junction-area is not enlarged, and on microscopical examination it is seen that the cartilage-cells of most of the epiphysis are quite normal, and that, instead of any undue proliferation near the line of ossification, as in rickets, there is really less than normal; the cartilage-cells at this level are large and spherical, but their arrangement in columns is very imperfect. The cartilage-cells are sometimes very small, and the hyaline matrix may present fibrillation but no calcification. The cutting off of the cartilage from the subjacent medulla by a layer of connective tissue affords an adequate explanation of the arrested growth in length of the long bones, and when the shortening is extreme it is probable either that the intensity of the disease was very severe, leading to much overgrowth of the connective tissue of the periosteum and its prolongations, or that morbid action was set up very early in fœtal life. The invading fibrous lamina between shaft and epiphysis, together with active development of periosteal bone below, will produce a great growth-pressure and so lead to yielding and curvature.

The *scapula* is often thickened near the edge of cartilage which may be partly overlapped by bone. This cupping, which as already mentioned also affects the limb-bones, may sometimes be well seen in the *ribs*, their anterior ends sending forward bony cups to invest the ends of the costal cartilages; similar cups may also be observed at the vertebral ends of the ribs. The anterior ones give rise to nodosities in the position of, but quite unlike, rickety beads. The ribs are also often short, thick, and heavy. The *innominate* bones are thickened, and frequently present the same overlapping of their epiphysial cartilages. The *heart* in two or three cases has been malformed,—open foramen ovale, stenosis of pulmonary artery, etc. With regard to other viscera nothing special has been discovered.

The most striking features in the morbid anatomy of sporadic cretinism are, first, arrested growth in length of bones which develop in cartilage, while the membrane-formed bones exhibit either normal or excessive growth; secondly, absence of the thyroid gland.

With regard to the bones, a study of their anatomy shows that the process is essentially the same in all. For it will be remembered that the greater part of a limb-bone (indeed, the whole of it, if we exclude metaplastic ossification: see article on Rickets) develops from vascular offshoots from the inner layer of the periosteum, and is, therefore, practically just as much a membrane-formed bone as one like the frontal. Now, in cretinism it is the fibrous lamina projecting from the periosteum between the cartilaginous epiphysis and shaft which hinders growth in length; there is no disease of the cartilage: its cells fail to proliferate and its matrix does not calcify in consequence, doubtless, of the vascular supply being cut off or diminished by the increase of connective tissue in the enclosing membrane. In cretinism, then, paradoxically enough, arrested growth in length of a bone is really due to increased growth of that factor, namely, the vascular periosteum which leads to the development of bone. Hence the shafts of the long bones and many of the cranial bones become thick and dense. Dwarfing of the limbs is present in most cases of cretinism, and the varying degrees of shortening are probably due to variations in the faulty development above described at the growing ends of the bones.

There are similar differences in the amount of change at the base of the skull, and, as already stated, no trace of disease, even in fœtal cases, may be visible. We must look elsewhere, then, for an explanation of the phenomena of cretinism; and this leads us to consider the condition of the thyroid gland.

Relation of Cretinism to Goitre.—Endemic goitre occurs in districts where endemic cretinism is absent, but wherever the latter is found the former is still more prevalent and extends over a wider area. About two-thirds of all cretins are goitrous, and in the remaining third large goitres are frequently met with in their non-cretinous brothers and sisters. It is stated that when both parents are goitrous for two generations in succession the offspring in the third generation are sure to be cretins. The association, then, is not accidental, but points to a common cause for the two diseases. To ascertain whether any closer relation exists between cretinism and goitre, we must needs study the condition and structure of the goitrous tumor. Here, as already mentioned, we get but little help from reports on endemic cretinism, but it is remarkable that in a large number of sporadic cretins no trace of the thyroid can be discovered, and it is probable that when the gland is congenitally wanting sporadic cretinism is always present. Further, the majority of goitrous tumors do not consist simply of an increased quantity of healthy tissue, but are cystic, fibro-cystic, or vascular diseases, in which there is more or less destruction of the glandular structure; hence in both endemic and sporadic cretinism, when a bronchocele is present, a microscopic examination is necessary before the existence or non-existence of healthy thyroid tissue can be asserted. It is reasonable to suppose that an old goitre will contain but little if any healthy gland-tissue, and a slowly progressing destruction of an organ may be expected to lead to the same phenomena as a complete absence of it; and that lack of the thyroid gives rise to a cretinoid condition has been abundantly proved by many recent experimental researches.

Cachexia Strumipriva.—In 1883, Prof. Kocher, of Bern, published an important paper on thyroidectomy and its sequelæ, in which he gave a graphic description of symptoms—in all respects resembling those of the remarkable condition first described by Sir William Gull under the term cretinoid, and since by Ord and others under the title myxœdema—which had almost invariably followed total extirpation of the thyroid gland. He points out that the relation of such a complex of symptoms to cretinism is obvious: in both there are arrested development, a large head, thick nose, enlarged lips, a coarse body, and hebetude of thought and speech. Reverdin, a few months before Kocher's communication, had also observed similar changes follow thyroideal ablation. These and the results of numerous other operations have been carefully analyzed and reported on by Dr. Semon, and they demonstrate that "in an important proportion of the cases" the removal of the thyroid gland in man is followed by the development of symptoms "exactly corresponding with those of myxœdema," and that when the operation is not followed by such symptoms the immunity is probably "due to the presence and subsequent development of accessory thyroid glands, or to accidentally incomplete removal, or to insufficiently long observation of the patients after operation." (See report on myxœdema in London Clinical Society's Transactions.) And in myxœdema itself occurring independently of operation, the thyroid gland is always found to be diminished in size, and to be undergoing a destructive change characterized by the substitution of a delicate fibrous tissue for the proper glandular structure.

Further, in experiments made upon animals, particularly on monkeys, extirpation of the thyroid gland has been followed by a train of symptoms closely resembling those of myxœdema and the cachexia of Kocher; and Horsley has clearly demonstrated that they directly depend on the removal of the gland, and do not result from injury to the trachea, to the cervical sympathetic, or to any other adjacent structures.

It seems probable, then, that sporadic and endemic cretinism, myxœdema, and the cachexia strumipriva are identical or closely-allied diseases, and dependent for the most part on diminution or loss of the function of the thyroid gland.

It is interesting to observe that both in myxœdema and in cretinism the ultimate cause, whatever that may be, *appears* to produce its greatest effect on connective-tissue elements. Their irritation and overgrowth are seen to be at the root of the bone-changes in cretinism as well as of the atrophy of the thyroid in myxœdema. The inconstancy of the degree of premature ossification in cretinism is explained partly by the period of life at which the morbid process begins, partly by the intensity of the latter, and partly possibly because in some cases the incidence of morbid action falls less on the membrane which develops bone than on connective-tissue elements in other parts of the body.

Prophylaxis.—In order to diminish the tendency to cretinism in endemic districts it is necessary—

1. To combat dampness of the soil and all other general causes of insalubrity, and to improve the hygienic conditions of the population in every possible way.

2. To make a careful selection of the available drinking-water, rejecting that which is much contaminated with earthy salts.

3. To treat promptly every case of goitre, either by removal from the

district, or, if this be impossible, by the administration of appropriate therapeutic measures, such as the external application of iodine, blisters, etc., and the internal administration of iodide of potassium or dilute fluoric acid; and in many cases operative interference will be desirable.

4. That mothers who have previously borne cretins should emigrate during the period of pregnancy to healthy places.

5. To send young children out to nurse in high and salubrious situations.

Attention may here be drawn to the fact that ordinary cases of sporadic goitre, or of exophthalmic goitre, or even the rarer instances of acute enlargement of the thyroid gland, may be followed by symptoms of myxœdema or the cretinoid state. All varieties, then, of goitre call for the most skilful treatment and demand our most careful supervision.

Treatment.—When fully developed, cretinism is incurable, but is capable of amelioration by suitable treatment: thus, the mental faculties may be developed by proper training in well-ordered asylums, and it is generally found that cretin children are less offensive and easier to manage and educate than other imbeciles. They should be kept as much as possible in the open air, and every means employed to strengthen the muscles and to improve the circulation. To this end systematic massage or proper gymnastic exercises, cold sponging, cod-liver oil, and the occasional administration of iron and other tonics often prove to be of great value.

THE URINARY DIATHESES:[1]

OXALURIA, PHOSPHATURIA, LITHURIA.

BY J. MILNER FOTHERGILL, M.D., M.R.C.P.

THIS article is an attempt to gather together what is known of an interesting subject not nearly so carefully studied now as it was half a century ago, and as it probably will be less than half a century hence. On the first two matters our knowledge is in a fragmentary condition, especially as to oxaluria. On the last subject we are in possession of considerable knowledge.

OXALURIA.

Oxalates appear in the urine as a slight cloud, closely resembling mucus. Sometimes glancing points can be detected in the urine as the light falls on a crystal of oxalate of lime. Such urine is of a pale straw or an amber color, the latter being the more frequent. Sometimes the oxalates cannot be found until the water has been passed a number of hours. The urine is usually of high specific gravity, and acid. Urea is present in good quantities, and uric acid and urates above what is normal. Quantities of phosphates are present, but are held in solution by the acidity of the urine. There is also an excess of mucus. Sometimes there is a certain vesical irritability, and heat and smarting are produced on voiding urine. Urea will break up into uric acid and oxalic acid. But how oxaluria comes about, is not yet known: it seems to have associations with nervous debility and imperfect or disordered digestion.

As to its occurrence in children very little seems known. Sir William Roberts makes several statements worth bearing in mind: "Every one who has had experience in calculous disorders cannot have failed to observe that the subjects of mulberry calculus, especially children, are not infrequently in the enjoyment of blooming health so long as no local irritation has been set up by the concretion." And again he continues: "Intense oxaluria may exist persistently without evoking the group of symptoms attributed to the oxalic diathesis. This group of symptoms may exist in typical development without the occurrence of deposits of oxalate of lime in the urine." This does not throw much light upon the subject. And as to its significance, he says, "At the most, it is only one in a long list of symptoms, and one of the least significant."

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[1] The manuscript of this article was received a few days after the cable announced the death of Dr. Fothergill. It was the last work of this distinguished writer.—EDITOR.

It is rather a matter of scientific curiosity, with its octahedral and dumb-bell crystals, than of clinical value, and some excellent works on diseases of children say nothing about oxaluria. The sort of child most likely to present it is that to be described at some length in the section on lithuria, and the reader will find that its associations are those of systemic debility. Two of Beneke's conclusions are of practical value:

1. "Oxaluria has its proximate cause in an imperfect metamorphosis,—*i.e.*, in an insufficient activity of the stage of oxidation which changes oxalic acid into carbonic acid."

2. "Oxalic acid has, if not its sole, its chief source in the azotized constituents of the blood and food: everything, therefore, which retards the metamorphosis of these constituents occasions oxaluria."

PHOSPHATURIA (WHITE GRAVEL).

Phosphoric acid is found in the body as phosphate of soda, a blood-salt of much value, and phosphate of potash, a constituent of muscle; and largely in the osseous system as phosphate of lime; while of phosphate of magnesia we know little beyond the triple phosphate in the urine. Phosphorus is a constituent of the brain-substance. Lecithin, a phosphorized fat, is largely found in the cerebro-spinal system. It is cast out of the body partly by the fæces and partly by the urine. In the urine it is found (1) as crystallized phosphate of lime, (2) as amorphous phosphate of lime, and (3) as the ammoniaco-magnesian phosphate (triple phosphate). This is about all that is positively known, and that phosphatic deposits are seen with alkaline urine. There seem considerable grounds for the opinion that phosphatic deposits are common with rachitis. All beyond that is opinion; and high authorities take different views. Prout held a "phosphatic diathesis" to be closely associated with nervous exhaustion; and the opinion is prevalent that when the nervous system is overtaxed phosphatic deposits are found. But Bence Jones called in question the phosphatic diathesis. It is exceedingly difficult to form any opinion on the matter. It is possible to look upon phosphaturia as the outcome of mal-assimilation. If the constructive operations are defective, the phosphates may be found in the urine instead of being deposited as bone. In the same way, if the liver is unequal to the construction of lecithin,—the phosphorized fat which seems to be the brain-food *par excellence*,—then the phosphates are found in the urine. Ordinarily the liver can break up phosphates for the phosphorus required for the brain; but in conditions of weakness it requires phosphorus in some less stable form, which it can break up. Just so in rickets we

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[1] Phosphates do not take up urine-pigments like urates. Hence the absence of color.

exhibit phosphorus otherwise than as phosphates. Then it is difficult to calculate the position as regards the phosphates in the urine. Their appearance may be due solely to the urine not being sufficiently acid to keep them in solution. If the urine could be made acid, would that do more than hide the phosphates? Suppose this could be done, would that affect the general health?—*i.e.*, if the phosphaturia could be proved to be due to some general dyscrasia? The matter bristles with difficulties.

The urine of the triple phosphate is copious, pale, and of low specific gravity. It is slightly acescent, and soon becomes alkaline. When this takes place before it has cooled, an iridescent pellicle forms on the surface, which really consists of crystallized triple phosphate, while tiny crystals attach themselves to the side of the test-tube. The sooner these alterations take place after the urine is passed, the more confirmed the condition. Constitutional irritability is associated with deposits of phosphate of lime. The urine containing this salt is not always pale, copious, and of low specific gravity, but may be quite the opposite. Still, such urine will become alkaline sooner than healthy urine. "When the urine is abundant and of low specific gravity, it is usually free from deposit; on being submitted to heat, however, it generally becomes turbid from a deposit of the phosphates. When voided in small quantity, on the contrary, the urine is often turbid when passed; and, in almost all instances, on standing for a time it deposits the mixed phosphates in abundance." (Prout.) Phosphate of lime may be amorphous or crystalline.

Phosphates become visible when the urine is rendered alkaline; vesical calculi become covered with phosphates when cystitis is set up; and calculi on section often show alternate layers of uric acid and phosphates. In the College of Surgeons of London is a specimen of which a section shows a lithic-acid core, then a covering of oxalate of lime, and finally an external coating of mixed phosphates.

From urine containing phosphates becoming turbid on the application of heat, a careless observer may (as some have done) mistake the cloud for albumen. As soon as the urine is rendered acid, the phosphatic cloud disappears.

A great deal has been written and said about phosphatic deposits, but we seem to know very little more than what we find in the urine, and its behavior; and this has more interest for the curious inquirer than practical value for the physician. The views promulgated by Prout have not stood the test of time.

Sir William Roberts says, "There is not the slightest reason to believe that there are any constitutional states specially characterized by an excessive excretion of phosphates." And when he makes a positive assertion of this kind, we all know he is fairly certain of his ground.

In fact, it would seem that, except so far as the urine and its examination are concerned, we know very little about either oxaluria or phosphaturia in children. And before we can get at that knowledge the urine must have left the body. The antecedent history is hidden from us. Yet that is what we should like to know. As regards lithuria, however, the case is widely different, as we shall see.

LITHURIA (THE URIC-ACID FORMATION).

"Children in general, and particularly the children of dyspeptic and gouty individuals, or who inherit a tendency to urinary affections, are exceedingly liable to crystallized lithic deposits from the urine." (Prout.)

This sentence may fittingly form a text for my dissertation. It is a sentence which may be thought over and pondered over with advantage by all physicians, especially physicians who see much of children in towns. Gout as "rich man's gout" which persons earn for themselves has taken such possession of men's minds, as regards the formation of uric or lithic acid, that "poor man's gout" is thrust into the background. Indeed, if it were not for the notorious frequency of vesical calculi in children, it might drop out of sight altogether. Yet I venture to think that the conditions under which we find lithiasis in children are such as to possess the highest interest for the reflecting physician. It is also very desirable that we survey the matter from its true stand-point. That alone can enable us to grasp the subject with a firm grip.

Lithogenesis is reversion.

When rudimentary kidneys appear in the animal economy, we find uric acid as the form of nitrogenized excretion. Up to the reptiles and the birds we find uric acid, except in the frog, which possesses a fluid urine containing urea. Animals with a solid urine void their nitrogenized materials in the form of uric acid. In the goose a certain small percentage of urea appears. When the mammalia are reached we find a fluid urine with the urine-solids mainly in the form of the soluble urea. Still, a small quantity of uric acid is to be found in the fluid urine, except in the herbivora, where hippuric acid takes its place. Even in man himself uric acid, in small proportion certainly, is found as a constituent in normal urine. Even the healthiest do not quite escape from their archaic inheritance.

Indeed, it would seem that man, at the threshold of life, commences with the uric-acid formation, which clings as a species of original sin, the tightest to the weakest. It is a well-known fact that uric-acid infarcts are found in the renal tubules of infants after the second day, and but rarely in the still-born,—a matter investigated by Virchow and others. A red powder is commonly found in the diapers of recently-born infants, which consists of uric acid and urates. This is not a morbid phenomenon, but "an undoubted physiological phenomenon," says Vogel; who, however, adds, "nevertheless it also furnishes cause for pathological conditions,"—a conclusion which is perfectly sound, as we shall see. These infarcts in the renal tubules in the newly-born are due "to the increased metamorphosis of tissue-elements which must take place after birth in consequence of the newly-inaugurated processes of digestion, respiration, and generation of heat." (Eustace Smith.) The fœtus is a reptile with the potentialities of a mammal. It has the circulation of the higher reptile; its heat-production is reptilian; and it manifests a tendency to the reptilian uric-acid formation. When it breathes, as by the touch of an enchanter's wand the circulation becomes that of the mammal, and its heat-production is that of a warm-blooded animal. Its other reptilian property, the uric-acid formation, is manifested most markedly in its early days of individual existence, and in healthy children gradually fades away to a shadow by puberty. A small proportion of insoluble uric acid can be held in solution, and so does no harm to kidneys constructed to excrete a soluble urine. But when it is present in larger quantity we find those "pathological phenomena" spoken of by Vogel. Sir Thomas Watson says, "Children up to the period of puberty are very liable to have lithic-acid gravel." Alison ("Pathology and Practice of Physic") writes, "Gravelly deposits of lithic acid and lithates take place frequently before the age of puberty," disappearing to return again as advanced life is being reached.

After the physiological process, it behooves us to consider the circumstances under which lithogenesis becomes a pathological condition. As it is a reversion to a primitive formation, we should expect it in feeble and delicate children rather than in the robust. It is indeed a *minus*, not a *plus*, quantity. It is not something added to a healthy child, but something taken away. The increase in the uric-acid formation is the measure of its shortcomings, its failure to attain to the normal urea formation. This is, to my mind, the proper way to look at lithogenesis. By so doing, many things are made clearer. We can more readily understand why the children of gouty individuals should manifest a strong tendency to urinary deposits and pass lithates. We also can comprehend why such lithatic deposits should be found in strumous children. Scrofula and struma are two words used to indicate a deteriorated constitution, a falling short of the normal physical perfection. (This matter must engage our attention more at length in a subsequent section.) In strumous children we should expect to find a distinct leaning to the primitive uric-acid formation. "The children of gouty individuals who have never themselves had gout in an open form are exceedingly liable to lithic-acid sediments. In certain modifications of the strumous diathesis, also, in which the tissues are of a loose and flabby texture, the deposition of lithic acid is very common. Indeed, the modification of the strumous diathesis when associated with gout, as is often the case, is perhaps more than any other condition of the system liable to lithic-acid deposits." (Prout.) Any cause, then, which acts injuriously upon the physique will bring about that deterioration to which we apply the terms scrofula and struma. Lugol, the great French authority, has gone into this matter with great care and ability,—so much so that his essay is a classical work. Among other observations he makes the following: "We could mention many large towns and cities where it is doubtful whether more than one in twenty of the indigenous population could be found entirely free from the scrofulous taint. The inhabitants of these towns are all scrofulous; those even who do not appear to be diseased are proved to be so, nevertheless, by the fact that they become the parents of scrofulous children." These observations fall in with my own experience. There is a certain deterioration of the physique in bred and born town-dwellers—which would readily take on scrofulous manifestations under given circumstances—in which the uric-acid formation is distinctly present.

In order to comprehend this matter, we must look at the effects of a town upbringing. The life of a large town is a life of perpetual excitement, from babyhood upward. The rustic child grows up with the pigs and cattle. Its existence is monotonous, and its brain-development slow. Not so the town child. What is the difference in their development? In the country child the three early layers of the embryo—(1) the epiblast, giving the cerebro-spinal system and the sensitive layer of the skin,—the means by which the organism is in communication with its environment; (2) the internal layer, the hypoblast, which furnishes the glandular elements of the digestive apparatus; and (3) the middle layer, or mesoblast, which furnishes the rest of the body-structures,—bones, muscles, and blood-vessels—all grow in fair proportion to one another. But in the town child the demands of the nervous system upon the mesoblast are such as to starve (to a certain extent) the hypoblast on the other side. In time these demands tell upon the mesoblast, with the result of a precocious creature with a dwarfed stature and feeble assimilative organs. These town products are to a certain extent an inferior race to their country cousins. One illustration of their inferiority is the tenacity with which they hold on to the early uric-acid formation. They do not outgrow it, like normal, healthy children. Even without presenting any outward signs of struma, they are moving in that direction. Many actually do present the features of struma in their finer form, the lofty brow, the long eyelashes, the tumid alæ nasi, the full upper lip—not necessarily having a chap in it; the bright little, precocious, angelic-looking children, whose delicacy of constitution is such that they rarely survive the ordeal of the exanthemata, and if they do not succumb to these maladies they perish by some tuberculous affection. These beings are deteriorating from the healthy standard in consequence of town life. My views fall in very much with those expressed by Lugol: "Scrofula shows itself in the children in the third generation of those whose ancestors entered Paris full of health and vigor, and from the third generation the malady rages even to the utter extinction of the family name." Scrofula will develop under one set of circumstances, while it tends to slight manifestations under more favorable conditions. "Latent scrofula is developed by debilitating influences in children who under more favorable circumstances would have escaped altogether." (Eustace Smith.)

"There are, however, other relations of the epiblast and hypoblast of the highest interest in this inquiry. That the tendency to lithogenesis may be acquired by the father and transmitted to his progeny is a notorious and well-recognized fact. Consequently, then, the causes of lithæmia in the parent are not to be ignored; especially the relations of mind and liver. The ancients spoke of *icterus ex motu animi;* and this view is still held by the vulgar in Germany. Indeed, some very eminent physicians of our time have been of this opinion. Not only are biliary disturbances induced by mental causes, but the other functions of the liver are not exempt. . . . Prolonged mental anxiety, worry, and incessant mental exertion not only interfere with the proper secretion of bile, but too often derange the process of sanguification and blood-changes, in which the liver is so deeply concerned, and induce lithæmia." (Murchison.) "That the condition of the mind has a powerful influence upon the manner in which the functions of the various organs of the body are performed, is at once rendered evident by watching its effect upon the digestive and renal organs." (Garrod.) Disturbance of the glycogenic function of the liver ending in diabetes is closely linked with mental worry. Disturbances in the other function, the metabolism of albuminoids and the oxidation of waste and surplus nitrogenized bodies, are also often, if indeed not mainly, of mental origin. The brain, as the organ of mind, powerfully influences the liver. "We are warranted in saying that the unexpressed emotion of anxiety, worry, and paralyzing misfortune, the grief unrelieved by tears, the load of care borne without help, the mind turned forever inward upon itself and checked in its active outgoings, even curtailed opportunities and soured ambitions,—that all such repression or want of expression by the usual channels is apt to take a peculiar revenge or to find a peculiar outlet by discharging itself unconsciously upon the glandular system, and upon the liver in particular." (Creighton.) Brain-toilers not only upset their own assimilative processes, but they beget children with what Drs. Budd and Murchison have called "insufficient" liver, who retain the uric-acid formation of early childhood into later days. Interstitial nephritis or chronic Bright's disease and diabetes are exceedingly common among male Jews, who are known to be hard brain-workers. Again, these two maladies are common—and, what is more, increasingly common—among men in the United States of America, who are recognized also to be hard workers. The wily Bengalee is saved by his dietary (he is no meat-eater) from Bright's disease, but he makes up for this by a still more marked tendency to diabetes. We see that hard brain-work not only injures the viscera of the individual, but also handicaps his offspring. The migrainous, lithogenetic daughter of the hard-working father is an object very familiar in my consulting-room. These workers predispose their children to lithiasis. Indeed, it would seem that a man shall not indulge in the luxury of amassing a fortune, on peril of begetting children, and especially daughters, with insufficient livers, to die prematurely of Bright's disease. As these men are now very common, especially in towns, they, their work, and its results, cannot be omitted from a consideration of the forces in action in keeping up the early uric-acid formation long after it is normally left off or outgrown.

This is a very serious matter, and its gravity must not be underestimated. The relations of epiblast and hypoblast are of the highest interest in connection with practical medicine. In the next section we shall see how far town life influences the organism in the direction of lithiasis; but it is well to precede this by some review of the men who take the direction of towns, and of the interaction of mind and liver. Anything which weakens the physique tends to rivet on the budding organism the uric-acid formation, from which it never escapes. And as town-born-and-bred children are now the majority, these imperfect beings call for our sympathy as well as our closest attention.

Etiology.—While robust children gradually outgrow and cast off the uric-acid formation of the newly-born, not so others. The offspring of the gouty and the strumous do not successfully escape from it as puberty is reached. Neither do the children of hard brain-workers, who have injured their assimilative processes by overwork; and what is acquired by the father is inherited by his progeny. Without necessarily presenting the features of struma, these children, and especially the female portion, possess a certain delicacy and sensitiveness. They are of mobile temperament and are emotional, and very often are charming little creatures. All recognize the bright, neat little town child, quite a little fairy as it flits about, presenting a strong contrast to the typical solid country child; but the latter is full of health and strength, while the town child is delicate and fragile. Medically these graceful and fascinating little personages are unsatisfactory. They are not all strumous, but they lean that way. They usually receive cruel treatment from those who least intend it. Bright, quick-witted, and affectionate, these mites are constantly amused and entertained when they would be much better left alone. I well remember one, the child of a distinguished American: two able and highly-intelligent women devoted themselves to it all day long. It had ducks and water-fowl in its bath, with which it played while the process of ablution was going on; and pretty it looked with its painted toys. But it never got far on its journey in life. It was easy to see what would happen, but by no means so easy to see how to help matters. The epiblastic nervous system makes severe demand upon the nutritive powers without such stimulations. The little fairy mite usually succumbs to the maladies of childhood, or is the victim of tubercular meningitis. The uric-acid formation is strongly marked in these delicate organisms. No wonder that Dr. Eustace Smith ranks "fear, grief, and other depressing passions of the mind" as among the factors which increase the tendency to lithogenesis in children.

These little organisms are sensitive and suffer sooner and more severely than more robust beings, if the drains are out of order. If exposed to the weather they are very liable to chills, which are followed by copious outputs of lithates. If they are confined to the house in bad weather, this tendency to form urates is encouraged. They are very liable to disturbances of the digestive organs, with acidity and flatulence. Kindly Dame

Nature tries to protect this delicate organism by a fastidious palate and a dainty appetite; but good-natured, blundering persons are always interfering, and trying to make it strong by feeding it up with beef tea and lean meat, which only further embarrass its feeble liver. They do not mean to do it harm,—far from it,—but injury is the sole result of their well-meant endeavors. Such children are found only in the houses of the opulent; they usually perish quickly in the homes of the humble. They are seen in the hospitals for children, and certainly in the out-patients' rooms of orthopædic institutions and in the children's wards of general hospitals. They are fragile creatures, usually with a light lower jaw and an arched palate, with strumoid features, and very commonly a diseased joint. The town-bred fairy is a hot-house plant,—an exotic, in fact,—which can exist only under very favorable circumstances.

Struma takes two forms. One is a bulky personage with the osseous framework of the large-limbed gouty individual,—of which Dr. Johnson, the lexicographer, is a well-marked specimen. Dr. Eustace Smith observes how pronounced is the uric-acid formation in these beings. They are degenerate forms of the gouty diathesis. But the neurotic, the person of the nervous temperament, even more readily degenerates into struma under unfavorable circumstances. And if any one will take the trouble to observe the children in the streets minus a limb, he will soon see how large a proportion are degenerate strumous neurotics.

Delicate, lithogenetic, neurotic children are not only dainty feeders, but they are also small drinkers. Again they are unfortunate, for the comparatively insoluble uric acid requires a considerable quantity of fluid to keep it in solution. Sir Alfred B. Garrod relates the case of a boy, under six, passing large quantities of lithates, who was much relieved by being induced to drink more freely: indeed, all persons who are the subjects of lithiasis should take a considerable quantity of fluid as a hygienic principle. Lithogenetic, neurotic children pass water which varies a great deal from time to time, sometimes a comparatively large bulk of low specific gravity,—certainly whenever under emotion,—and at other times a scanty dense urine soon becoming turbid and throwing down a copious sediment. Their bladder is a source of much trouble to them, and its calls are often peremptory. They suffer a great deal when travelling, especially in countries where the English system of railway-carriages obtains. These are the children who at school after any trifling emotion quickly ask "to go out."

The late Dr. Bence Jones classed lithiasis among the "Diseases of Sub-oxidation," in which he kept an important matter to the front. Urea is more highly oxidized than uric acid, and therefore the matter of the supply of oxygen is very important in dealing with lithogenesis. We all know how beneficial to strumous children is country air, especially by the sea-side. Some of us are aware how badly neurotic females born in the country bear confinement in towns. They actually pine for a breath of fresh air before many months are over. And what is the difference between the fresh air of the country and the air of towns? The fresh air contains oxygen in active form, known as ozone; while repeated observations have never been able to find ozone in the air in the middle of towns. No wonder, then, the sensitive lithogenetic beings, suffering under a form of suboxidation, find town air so little to their taste, and are so fond of excursions to the country. Fresh air is favorable to the healthy urea-formation; and, though they do not, of course, know why, they recognize the fact readily enough that they are all the better for being in the country. And, what is more, these subjects of lithiasis do best in bracing localities, and are not so well in low-lying relaxing places. Just so, too, the bilious; and the bilious young person often becomes gouty at a later period of life. It would seem that the liver requires plenty of oxygen in order to carry on its operations properly. Country children, spending a large portion of the day in the open air, are comparatively free from these diseases of suboxidation which afflict town children, whose days are largely spent in-doors and who do not breathe a very pure or salubrious atmosphere when out in the streets. Looking at gout as a disease of suboxidation, Dr. Bence Jones regarded an acute attack of gout in an old gouty joint as an oxidizing process carried on by means of the fuller blood-supply of inflammation. And certainly such attacks are cleansing processes.

In enumerating the causes which encourage and foster the continuance of the uric-acid formation, we must bear in mind the food-customs of to-day. We all know how "rich man's gout" is the result of indulgence beyond the body-needs in food and drink on the part of the individual or his ancestors. The plebeian alderman often eats and drinks with impunity, but leaves gout behind him as part of the inheritance of his children. Very commonly, as age advances, he also makes the acquaintance of gout himself. Alison tells how lithogenesis belongs to childhood, disappearing at puberty, to reappear again later on in life,—a matter illustrating the old adage "once a man and twice a child." We all know how good living bears on this reversion to the primitive uric-acid formation, and the effects of a lack of exercise. And we also know how, by temperance, the gouty man can keep his foe at bay. Bearing all this in mind, we can realize how an injudicious dietary can handicap the growing organism and prevent its escape from the primitive uric-acid formation. The prevalent practice of "feeding up" delicate children is at once irrational and pernicious.

If the Creator has decreed that certain children, procreated and born under certain circumstances, must be inferior organisms to normal children, who come into the world under more favorable circumstances, we had better recognize the fact, and bow to it. Looking at the matter in its proper light, it is nothing less than wicked and cruel to attempt to "feed up" these poor mites. When the writer was the senior resident medical officer to the Leeds Public Dispensary (1870–71), he saw numerous instances of the evil effects of giving meat—*i.e.*, animal food—to young children. Again and again babies of a tender age were brought there with the genito-urinary organs all scalded and raw from the irritant character of their urine. On inquiry it constantly turned out that the fond father was in the habitual practice of giving the infant part of the meat prepared for himself. On discontinuing the baneful practice the child soon got all right, with the help of a little potash. The lesson then learned has not been forgotten, and the writer often reprehends the crazy practice of trying to make a weak child a strong one by giving it meat in liberal quantities,—which only makes it worse and, feebler. Strong meat is not for babes. Beef tea is also injurious when taken freely. It contains nothing that can feed or nourish the body, though popular opinion credits it with marvellous virtues. Its constituents are past the stage of albumen for tissue-building; its kreatin and kreatinin are at the head of the descending series which ends in uric acid and urea. And its advocates must remember that, while it is not a food, it can add to the load of uric acid, whose burden is already a tax upon the system. Again and again has it fallen to the writer's lot to see lithates appear after a course of strong beef tea, and even attacks of articular gout in lithogenetic neurotics.

The diet of the nursery laid down by the wisdom of the ages—viz., milk and farinaceous matters—is the proper food for infants. And if delicate children cannot digest farinaceous matters, it is easy to supply predigested "foods," which will be found to give infinitely better results than the prevalent plan of giving meat and meat infusions, to the poor child's detriment.

The unfortunate child will find it hard enough work to escape from the thraldom of the uric-acid formation without its fetters being riveted upon it.

Diagnosis.—In my student days beakers containing a specimen of each patient's urine were a prominent feature in the wards of hospitals. Gradually a test-tube containing some urine which had been tested for albumen took the place of the beaker, until the latter has well-nigh disappeared. It has recently reappeared in my wards, and this example will be followed elsewhere before long as the relations and associations of lithiasis become more completely realized. A deposit of lithates tells of a lower urinary formation, and is a "storm-signal" whose value will depend largely upon the knowledge of the individual observing it. Prout regarded urates as a sign of evil omen in organic disease. When steadily present in cases of pulmonary phthisis, they are the heralds of disaster, in my experience. But this is scarcely the diagnosis of lithiasis. Of course the diagnosis is made by allowing the urine to stand overnight in a cool place, and then examining it by first inspecting it. It will often be found to contain a deposit. First ascertaining if it be acid, and finding it so, the character and appearance of the deposit call for our attention. (I do not conceive that the scope of this article extends to an elaborate examination of urinary deposits, and therefore will take just so much of this part of the subject as pertains to the matter in hand.) Urate of soda forms a white or yellowish deposit, which sinks readily. It is very common in children when they have caught a chill or a cold. Or it may appear in pyrexial maladies. Sometimes the urine is turbid when voided. Especially is this the case in strumous children. Sometimes the spiny crystals cause great irritation in the urinary passages. But hæmaturia is less common with children than might be supposed. Urate of ammonia is also pale, but is not deposited until the urine becomes ammoniacal. The fawn, orange, brick-dust, pink, or deep-red deposits are the amorphous urates. Uric acid gives the well-known "cayenne grains" or brown crystals; but sometimes the crystals are so fine as to simulate the deposits of amorphous urates. "Urine depositing uric acid has commonly a rich yellow or orange color, and is invariably acid." (Sir William Roberts.) Uric acid is highly insoluble, and so are the urates; consequently, when the urine is scanty the deposit is comparatively copious. Children presenting strong evidences of the uric-acid formation usually pass either a large bulk of urine, pale and clear, of low specific gravity, or a denser urine giving a considerable deposit. And these frequently alternate. When the urine is turbid when passed, it contains urate of soda, which falls on cooling. Urate of soda forms crystals and concretions within the body. These may form in the tubules of the kidney and remain there, or fall into the pelvis of the kidney and lodge there, or find their way into the bladder. Lumbar pain is felt when the stone is in the kidney. Contrary to what might be expected, renal crystals do not usually produce a bloody urine in children. When the blood comes from the kidney, it is thoroughly mixed with the urine,—bloody urine; while hemorrhage elsewhere gives blood and urine. When the concretion is in the bladder, the child will pinch and pull its prepuce. It will cry with pain on emptying the bladder, while sometimes the stream of urine is suddenly arrested by the concretion blocking the outlet. Violent exercise causes pain; and the late Mr. Teevan used to say that a ready method of deciding whether it was desirable to pass a sound or not, was to get the child to jump off a chair: if it was ready to do it a second time, it was highly improbable that any stone was in the bladder. Common as is stone in the bladder in children, it is really a rare outcome of the uric-acid formation, especially in girls. For anatomical reasons, girls scarcely ever have vesical calculi; yet lithogenesis is very frequent with girls.

Wetting the bed at night has close relations with uric acid, and in all cases of nocturnal incontinence the urine should be examined. In my experience, wetting the bed occurs mainly in two classes of children,—in very bright, vivacious, neurotic little girls, and in comparatively dull and backward children of low nervous organization. There is a heightened nervous susceptibility in one case, and a defective condition in the other. In either case the uric acid present plays a part. The purely neurotic child is the sprightly little fairy described in the last section. Something may now be said about the strumous child with lithogenetic tendencies. If it is the weakly organism which never effectually and satisfactorily outgrows the uric-acid formation of early life, then of course the strumous will suffer. Struma or scrofula is generally a degraded organism, with tissue-inferiority to a greater or less extent. There are forms of struma which give beautiful children, delicate bright creatures, as well as forms of it which give plain features and uncouth figures. But, be they fair or ugly, there are outward indications which accompany the uric-acid formation of liver-inability, with which it is well to be familiar. Looked at as defective organizations, they possess an interest of their own. Lugol describes them so exactly that a quotation seems to me to be desirable: "The scrofulous habit, although it is in general characterized by indolence and apathy, is not altogether incompatible with a certain amount of bodily activity; this very activity, however, instead of tending to the increase of the physical strength and development, as in healthy subjects, on the contrary assists in diminishing its powers: we observe, therefore, that scrofulous children in whom this more than usual activity is manifested are quickly fatigued, and are slow in repairing their exhaustion. The genital organs of scrofulous subjects are generally more or less retarded in their development, and seldom acquire the vigor which characterizes a healthily-constituted individual; young men eighteen years of age, or even older, are often in this respect little more advanced than children of eight or nine years. In some cases one testicle only is found to have descended at the age of twenty years, and occasionally both have remained in the abdomen. Young females are no less backward in their development than the other sex, often presenting no signs of puberty at the age of eighteen years. Menstruation is not established without the concomitant of dysmenorrhœa, which lasts for two or three years, and in some cases for their whole life. The menstrual discharge seldom possesses healthy qualities; it is either insufficient, of only one or two days' duration, or excessive, lasting six or seven days; in neither case does it produce satisfactory effects upon the economy, for it is not accompanied by the other signs of puberty."

This raises a question of high interest in the relation of the uric-acid formation with defective or imperfect organisms. The imperfect development of strumous beings has been observed by others than Lugol. "Most scrofulous persons are of small stature and have slender limbs; nor is it very uncommon in such individuals to find some member or organ imperfectly developed, defective in its power, or curtailed of its proportions." (Cumin.) Strumous beings have imperfect reproductive organs, as a rule. If an organism is imperfect, we should *a priori* expect to find organs which remain infantile for a considerable period of years, and whose development marks off the child from the budding adult, to feel the general backwardness most. Even if the external portion of the sexual apparatus develops, the internal organs remain infantile in girls; and that, too, not only in the distinctly strumous, but also in the neurotic girls so common in towns, who are somewhat degenerate, but not so distinctly so as the strumous. A great many slight girls, especially town products, exhibit the same imperfect condition of the reproductive organs as the strumous. Some are sexless. Some are feebly erotic. Some never menstruate. Some menstruate, but the menses are scanty and accompanied by much suffering. When they marry, some are sterile, and the slighter and more bird-like the creature the more certain is she to be childless. Some bear one or even two children so delicate that they cannot be reared. It seems, indeed, that Nature has laid her plans to keep up the race from the strong.

To show how closely related are the neurotic to the strumous, I may, adduce three sisters, patients of mine at Victoria Park Hospital. Their mother is a healthy-looking woman, born and brought up in the country. The father, also of country bringing-up, is reported to be strong and healthy. But, for some reason or other, their progeny are distinctly defective. The eldest daughter, now sixteen, is slight, with the short ungainly figure of the strumous,—with stubby nose, tumid upper lip, and ill-cut features like a blurred photograph. She presents no signs of puberty. The second, now fourteen and a half, has sharply-cut features, with a slight physique, and talipes valgus in both feet. She has been treated surgically, with unsatisfactory results. The third, now thirteen, has also sharply-cut features and a very slight physique. She was a small, backward child, but at twelve shot up wonderfully. She is a migrainous neurotic, with heart-troubles and digestive troubles already well marked. Poor little mite, with her uric-acid formation and her narrow chest, it will not be very long before chronic Bright's disease will dig her grave and pulmonary phthisis will bury her! While the eldest in figure and feature is distinctly strumous or scrofulous, these terms could in no way be applied to the two younger girls. They are slight neurotics, small in the bone and light in weight. But the family illustrates very clearly how little is the gulf betwixt delicate neurotics and the actually strumous. Possibly the circumstances of the parents improved anterior to the procreation of the two younger girls. This matter calls up a family who were my patients years ago when in general practice in the North of England. The father was a slight, neurotic man; the mother distinctly strumous. Their first two children were healthy. Then came illness in the father, and, with that, poverty. Two children born during this time were distinctly strumous. Then the father came in for a small income, and food was no longer hard to procure. Two more children were born after this, and, like the two eldest, could not be designated strumous. Physical degeneracy is a complex subject; but sure it is, physical degeneracy is wedded to the uric-acid formation, and divorce seems impossible. Wherever and from whatever cause the physical development is thwarted, the organism is prevented from outgrowing the lithogenesis with which the human frame makes its start in life. Lithuria is the brand of physical inferiority.

Not only may vesical calculi be found in young children, and lithatic deposits, especially after a cold, but infants may have outputs of gravel,—red sand.

Dr. Debout d'Estrée, of Contrexeville, informs me that gravel is far from infrequent with very young children, many of whom are brought there for the benefit of the water. Actual gout—true articular gout—is not unknown among children who have not entered their teens. One case is reported to me where a youth under twelve had an acute attack of gout in both great toes simultaneously. One lady patient of mine, now seventy years of age, told me how she was plagued with articular gout in her youth, which entirely disappeared when she was twenty, and has never returned. It was a strange story, but her husband vouched for the accuracy of the statement, and the hands still, after fifty years, corroborated her tale. This surely was a case of failure to outgrow the primitive uric-acid formation till a late period. I may add, the pair were childless. Failure to outgrow lithogenesis must clearly be differentiated from the gout of later years brought about by good living, when a competent liver which has escaped from the early uric-acid formation reverts to it because it is wearing out under the burden imposed upon it.

As to making sure that sediment precipitated in urine is really uric acid, the whole can be stirred up and some of the turbid fluid be placed in a test-tube. Usually, on heating, the urine becomes quite clear. Or a drop or two of liquor potassæ can be added to another specimen, when the turbidity disappears. (This last gives the observer a very good conception of the action, within the body, of potash as a uric-acid solvent.) Or some potash can be added, and then heat applied. Or a solution of borax or phosphate of soda can be used. The most sensitive test is to place a drop or two of the urine on a slip of glass, with a drop of strong nitric acid, and place it over a spirit-lamp until it dries into a yellow residue. When cold, touch this residue with caustic ammonia, and the characteristic bright-violet hue (murexid) is instantly produced.

As to the qualitative analysis of uric acid, it is in an unsatisfactory state. We know very little yet, if anything at all, as to why and under what circumstances uric acid is retained in the body, and how it comes to be cast out at times in large quantity.

There are also definite appearances under the microscope, for those who have leisure and wish to study the subject.

The urine of persons of the lithic diathesis "is more acid than the urine of health, and gives to litmus-paper a deeper shade of red," says Sir Thomas Watson, who continues, "The presence of this diathesis is likewise accompanied by, and so far denoted by, the tendency to fevers and inflammatory complaints." The readiness with which lithæmic children catch cold has been spoken of before, in connection with the diminished resisting power of persons of this lithic diathesis. Such colds are always productive of a large output of lithates. The question may be raised as to how far the excess of lithates present in the body is a predisposing cause, and a reason why the child catches cold on slight exposure at one time and escapes at another. When fairly rid of their lithates, these children seem better and happier,—as a strumous child at the sea-side, for instance. Children of a larger growth who pass quantities of urates always are better in the country than in towns; and when for any reason they become town-dwellers, they require visits at repeated intervals into the country to keep them in fair health. The question of the relation of lithiasis to acute rheumatism in children is one well worth investigation; for their urine is at that time highly charged with lithates. The subject is one on which, evidently, we have still much to learn,—and much, too, that will have a high practical value as towns enlarge and more and more children breathe an air deficient in ozone.

Pathology.—The kidneys of the young do not suffer from the passage of lithates as do those of lithæmic persons of maturer age. If the uric-acid formation is normal at the outset of life, this will occasion no surprise. The kidneys will be adapted to their work. With lithiasis in later life interstitial nephritis is the rule; with the lithogenesis of early life such change is the rare exception.

Probably, too, it is pretty safe to make the broad statement that the viscera of the young are not so prone to chronic interstitial changes as are those of later life. Be this as it may, interstitial nephritis is not a disease of child-life. When attending the Pathological Institute in Vienna, the kidneys of a child of eight presented very distinct evidence of this change, while many young adults had kidneys extensively diseased. My colleague Dr. Eustace Smith records a case where a child of twenty-one months had only one kidney, and where "the capsule was adherent, and, on removing it, a small portion of the renal substance was torn away with it. The surface of the organ was very granular and irregular." In the young, then, it may be said that the kidneys do not, as a rule, suffer from the output of lithates in comparatively large quantities. But when the system fails to cast off or outgrow the uric-acid formation,—to the normal extent, at least, —how about the kidneys then? This is a widely-different matter, there is every reason to believe.

Our knowledge of changes in the circulation and the kidneys of young lithogenetic adults is far beneath our acquaintance with such morbid phenomena in persons of advanced life, where the liver, from some cause or other,—from overfeeding up to overworking mentally,—fails in its urea-formation and reverts to the lithogenesis of early life. Vaso-renal change is the shortening of days, as Dr. Goodhart says, and very happily too: "Old age is not an entity, but a set of conditions predisposing to that state which is called chronic Bright's disease. And though to most this comes in the natural order when the prime of life is run, yet to some old age is no matter of years and of averages, but the running down of a spring set for an individual." And it seems to me that with the lithic diathesis the spring runs down quickly, sometimes very quickly. In order, however, to take a firm hold of the matter, it may be well to review the morbid phenomena of the vaso-renal change. Lithiasis in the robust of frame runs on the old well-recognized lines of gout,—articular gout, visible to the eye, recurrent attacks of bronchitis, which lead to emphysema, with a large heart, which is liable to cause disease in the valvular apparatus. (When the valve-mechanism of the higher heart is injured, compensatory hypertrophy follows,—*i.e.*, an increase in the bulk of the primitive muscular sac,—the lower heart. But when from any cause the muscular wall waxes out of proportion to the valves of the higher heart, these are apt to become affected from the additional strain thrown upon them.) These are the changes manifested by persons of the Norse type. But with the slighter neurotic individual the morbid phenomena are widely different. Something has already been said about the excessive demands of the epiblast leading to a comparative starving of the hypoblast. So, in the neurotic person of the uric-acid formation, we find digestion-troubles to take the lead. There is indigestion, with acidity and flatulence often alternating, and more or less constipation. Very often, too, there are hepatic derangements. The epiblast itself suffers, and the epidermis is liable to herpes; while sometimes there are eczema and affections of the true dermis. There are migraine and other neuralgic affections. The heart is liable to palpitation, and to the opposite condition of failure, resembling syncope, but too frequently without loss of consciousness. The mind, too, has its characteristics. It is acute and clear in some, wayward and flighty in others, while elation and depression alternate,—the child is "all up or down," in common parlance. The mood is variable; pretty irritable, as a rule, especially when hemicrania is present. All readers of experience will recognize the outline, and be able to fill it up. Not only women but also many neurotic men present these linked phenomena. And it is among these persons that we are apt to find "pure cussedness" to prevail. They usually have plenty of good sense, but they are not always in the mood to exercise it.

Like the Norse type, neurotic persons of the uric-acid formation present certain changes in the vascular system.[1] Uric acid is comparatively insoluble, and the self-preservative power of the system increases the bulk of the urine by tightening the arterioles upon their contents and so increasing the watery constituents of the urine. The left ventricle has more resistance to overcome on systole and hypertrophies; while the high blood-pressure in the arterial system leads to that hardening process known as "the atheromatous change." The vascular system thus modified is liable to aneurism and apoplexy, as well as to palpitation. Further, passing disturbances of the vaso-motor nerves give rise to angina pectoris vaso-motoria. And it is in neurotic persons of this lithic diathesis that we find angina, as a rule, while the kidneys suffer. However tolerant of uric acid in early years, this tolerance passes away in time. When the uric-acid formation is pronounced, either the uric acid remains in the system, giving rise to various gouty phenomena, or the kidneys are injured by the constant output of lithates, with the result of interstitial nephritis. Commonly enough both are found together. A considerable injury or mutilation takes place before the condition of the kidney is forced upon our attention. Says Prout, "It may be well to remind the reader that the tendency to lithic-acid deposits is almost invariably connected with an hœmotrophied[1] condition of the kidney." Sir A. Baring Garrod remarks, "I have met with numerous instances of the occurrence of gout and calculus in the same individual, but with few in which they were present at the same time: it is not at all uncommon, when taking the history of gouty patients, to find that when young they had suffered from calculi, and that gout supervened at a much more advanced age." And Sir William Roberts says, in connection with lithiasis, "The kidneys themselves suffer: their secreting tubules and the interstitial substance are clogged with urate-deposits, which constitutes one of the most fatal forms of chronic Bright's disease."

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[1] Certain readers may say this is wandering from the subject in hand. To this I demur. It is often necessary to read the parents, in order to see the probable future of the child and give it true help.

Such, then, is the march of disease when the system either is unequal to casting off the lithic-acid formation or reverts to it again at a later period of life. As to young persons, it certainly shortens the span of life. Some time ago I was called in to see a slim woman of thirty-three who had for some time been the subject of gout: she had a large heart, with hardening arteries, and a cerebral artery had snapped. Her system was truly senile, and the spring ran down in half the ordinary time. One of my hospital out-patients, a comparatively large, strumous girl of seventeen, passes urine loaded with lithates. She, too, has a large heart and hardening arteries, has cardiac and digestive troubles, and, in all human probability, shrinking kidneys. Further, the slightly-built girl of thirteen, mentioned in the last section, manifests these morbid phenomena distinctly. To make sure that my imagination was not getting the better of my clinical acumen, one day I called in my then colleague Dr. Angel Money, who quite agreed with me as to the actual existence of the morbid changes. Her spring, I fear, will run down before she is twenty-one. One day a youth of fourteen was brought to me by his father, a medical man, and, on examination, abnormal dulness was found in the region of the liver. Thinking my colleague Dr. Heron would like to see the case, we went to his residence. We could come to no conclusion as to the dulness, but both thought the boy had a renal aspect. Some urine was passed and tested for albumen. No albumen was detected; but Dr. Heron, later, informed me that the urine after standing was the most highly charged with lithates he ever saw.

The matters of the pathology of the vaso-renal changes, with their consequences, started up by the retention of the lithic formation after the days of childhood are passed, are of great importance as regards prognosis and treatment, which remain to be discussed.

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[1] Hœmotrophied—cirrhosed.

Prognosis.—The lithic diathesis is linked with physical inferiority,—as a broad rule, at least. As lithates are normal at a very early period of life, all children, weak or robust, will pass lithic deposits more or less. But while the robust soon get away from the primitive uric-acid formation, not so the weakly. To them it clings, and certainly seems to predispose them to colds and chills, and even inflammatory affections. We are familiar with the secondary inflammations of renal inadequacy in advanced life; and possibly we may come to find a somewhat similar relation betwixt the lithic diathesis and inflammatory affections in childhood. Not only are such children more liable to inflammatory affections, but they also manifest less resisting power. When in general practice, it always seemed to me that the children of the dyspeptic and lithæmic were specially liable to go down under bronchitis. This may be no more than an impression remaining on my mind; but, from the foregoing consideration, it would seem not improbable that the impression is correct in the main. These children are more difficult to rear than other children, and more apt to die under the searching test of disease than more robust children. Before the day of the test-tube the urine was examined by the eye, and almost by it only. The presence of visible deposits had then a significance not given to it today, when the urine is examined for albumen and sugar and little else. But if the foregoing consideration is at all correct, it would seem that the significance of urinary deposits, especially lithatic deposits, will ere long be again recognized as important. If these deposits are found at and after puberty, they are highly instructive, diagnostically and prognostically.

In the summer of 1887 a tall, slight girl of fourteen, but looking more like sixteen, came into my wards with localized pneumonia at the base of the right lung. The disturbance of the general health was slight, the fever was not high, and there was no reason, apparently, why the case should not run the usual course to a satisfactory termination. But, instead of improving, the case seemingly came to a stand-still. After a time the girl grew worse, and the disease-area extended. Then it was observed that her urine threw down copious lithates. The subject of the prognostic significance of lithates in the course of phthisis is a topic on which I occasionally dilated at the hospital, and it was suggested to a recent resident medical officer as a subject well worth investigation and likely to give results of a practical value. But it never was taken up. Still, it had not escaped my mind, and, as the deposit continued and the case steadily got worse, directions were given to Dr. Sidney Martin, the pathologist, to make a note as to the condition of the kidneys. When the opportunity came, one kidney was found fibrous and cystic. Yet no albuminuria was found in life. But there were lithatic deposits which had aroused suspicion. After that the kidneys were examined systematically; and already Dr. Martin has collected quite a little series where interstitial nephritis has been found coexistent with lung-mischief, if not pre-existent to it. The next case which came under my personal notice was that of a young man with localized mischief in his right lung-apex. The disease did not extend, nor were the symptoms acute. Still, he did not make headway. He, too, had urinary deposits. Soon it was apparent that he improved and got worse again in cycles of about a week. He would brighten up and take his food for a day or two. Then his appetite fell off, and for a day or two he was very miserable. Then would come a distinct output of lithates, after which he would brighten up for a time. It was evident that there was a factor in the case which handicapped the poor fellow; and it seemed to me there must be some mischief in the kidneys, though, of course, of what kind it was impossible to say. After each cycle he was perceptibly worse, and before long he sank. The lung-mischief, which had been obscure, was found to be disseminated tiny bronchiectases, which certainly presented a phthisical aspect. Both kidneys were found to be extensively fatty,—a degenerate form of interstitial nephritis.

When attending the Pathological Institute in Vienna, my mind was much exercised by the exceeding prevalence of chronic renal mischief, both granular and fatty. My impressions at that time (1871–72) were that chronic interstitial nephritis was a disease of middle age and advanced life and closely linked with gout; that it was, indeed, a disease of protracted good living. Yet these Viennese subjects were many little more than twenty, and largely from twenty to thirty. Too much good living scarcely came their way. Bread and potatoes and beer of a very light character were the staple of their food. There was no nitrogenized excess in such a dietary. The matter has often exercised my mind since, but it has only been of late that light has dawned upon it. So long as the unwise habits and food-customs of the individual held the field as the causal associations of the granular, cirrhotic, or gouty kidney, so long light was not likely to come. But the further study of "poor man's gout" began to show that interstitial nephritis was the lot of sundry individuals who were sparing eaters, but who still remained under the thraldom of the uric-acid formation of an early period.

My observations at Victoria Park Hospital have thrown some light upon another matter which struck me greatly in Vienna; and that was, how readily the poor Viennese sank under their maladies. Cardiac valvulitis frequently led to death, with the dropsy and serous effusions of the failing heart, in a period of fifteen months,—a matter quite opposed to my experience at the Leeds Public Dispensary. In other diseases the same readiness to die was manifested. That the conditions of life in Vienna were scarcely compatible with a robust constitution, especially among the poorer classes, was patent enough to a person of any experience. But the Yorkshire folk did not die easily like the Viennese. Now the matter has become fairly plain. If the retention of the early uric-acid formation beyond puberty is an evidence of tissue-inferiority, and of an insufficient or incompetent liver, if the prolonged passage of urates in excess through the kidneys inflicts injury upon their structures, then it is easy to understand how an organism so handicapped readily gives way under the trial of disease.

Another matter is worth keeping in mind in connection with this topic. The lithatic neurotics as well as the strumous have imperfect digestive organs. They suffer the pains of indigestion on slight provocation. Pastry of all kinds, which forms a large portion of the dietary of country-people who can digest it, is beyond their powers. Fat, especially as a piece of animal fat, they loathe. Cream is beyond the means of most. Cod-liver oil is not palatable. Consequently they live to a large extent on the flesh of animals—so far as they can purchase it—and bread, toasted or fried; the sapid meat is to their taste, and does not give them the stomach-ache. Unconsciously these victims of the lithic-acid formation are pushed along the downward path by their digestive incapacity. Of imperfect assimilative powers, with an exacting stomach, and no free oxygen to help the liver in its straits, what may we expect? Just what happens. Bright's disease and pulmonary phthisis are the scourges of degenerate and degenerating town populations. Commonly they are found together. Dame Nature, merciless and unsparing, weeds out the weakest. No plea of extenuating circumstances is of any avail with her. In one matter only is she compassionate: she makes the females sterile. Those organisms which are too feeble to throw off the uric-acid formation do not usually attain full and complete sexual development; and so the degenerating process ends.

Treatment.—When we see a fire choked with its own ash, we do not throw on more fuel, but stir the fire, so as to get rid of the ash and admit the air to the dying embers. The fire cannot get on, for want of oxygen. Just so the delicate organisms of the uric-acid formation. They also are embarrassed with the incombustible ash of the body,—the nitrogenous waste. Remove that, and the organism feels relieved from a burden that was weighing it down. All compounds in which nitrogen is a factor oxidize with difficulty, and, according to Liebig, the presence of nitrogen interferes with oxidation. Consequently we can see how the nitrogenized waste is the incombustible ash of the body.

The first step to be taken is, then, to promote oxidation. We all know how children of the lithic diathesis, as a strumous child with a diseased joint, picks up and thrives when at the sea-side. Even a few hours by the sea will do perceptible good, one recognized authority on children's diseases assures me. The free oxygen helps the liver to keep up the urea-formation. Indeed, as lithiasis is a disease of suboxidation, plenty of active oxygen is what is required to remedy the condition. Such a child should be reared in the country if possible. Parental affection stands in the way; but, in fact, kindergarten schools by the sea-side are a matter of the future. It will not, however, be possible to overcome the natural feeling of parents to wish to see their children around them,—and especially those fairy mites, the strumous neurotics, with their winning ways,—until the realization of the pernicious effects of town air upon growing organisms has sunk deep into their hearts. A thousand difficulties and objections stand in the way, and can be overcome only by a distinct consciousness that it is but simple duty to the town child to place it in a more favorable and less injurious environment.

And that day is not yet near at hand. "A child with a scrofulous diathesis should learn its lessons in the fields, and not be bound down to books in the crowded atmosphere of a school-room." (Cyclopædia of Practical Medicine.)

That is a good, wise, kindly, sensible way of putting the child's necessities. Kindergarten teaching, in the open air whenever the weather will permit of it, must become a general practice. What says Dr. Eustace Smith anent this matter? "Too close confinement to the house, especially in cold, damp weather, in some subjects, loads the urine with uric acid or its compounds."

That is the outcome of confinement in an impure atmosphere upon these sensitive organisms, which feel so keenly what scarcely affects others. The long yearly sojourn by the sea-side or among the mountains is no longer a luxury for town-dwellers' families; it has become an imperative necessity, essential to health, as towns grow larger. Plenty of fresh air is the first essential.

"The treatment of the lithic-acid diathesis must be directed to the removal of the prime causes of this condition,—viz., mal-assimilation, defective oxygenation of the blood, and the ingestion of too large a quantity of stimulative food." (Erichsen.) Mal-assimilation is the starting-point. The defective organism is defective because its assimilative organs lack power. The system fails to outgrow or rise above the uric-acid formation which is normal at the threshold of life. Bearing this well in mind, we must see that it is not wisdom to overload these feeble viscera "under the impression that strong food is necessary to give the patient strength." (Erichsen.)

It is not what is swallowed, but what is assimilated, that nourishes. This sentence might with advantage be written up over the nursery door. What says Lauder Brunton on the feeding up of the weakly? "What does the patient say when he goes to his medical attendant to describe his case? 'I take all kinds of strengthening things, and yet I feel so weak.' If, instead of using these words, he were to say, '*Because* I take all sorts of strengthening things, I feel so weak,' he would express a part, at least, of the truth." This puts the matter in a nutshell. Dame Nature has her kindly moods, and, when sending a child into the world with an insufficient liver, protects this feeble viscus by endowing the child with a small appetite. It is a small, dainty, fastidious feeder, much to the chagrin of its nurse. Its food must be served up with scrupulous cleanliness and neatness, otherwise its appetite takes wing on the spot. It grows up with the lithic-acid formation, and often biliousness; for both alike are the outcome of a feeble liver. Vain are all attempts to feed it up; it simply cannot be fed up. Blundering busybodies do it harm rather than good by stepping in and traversing nature's arrangements. The appetite keeps guard over the liver. When the liver is embarrassed, the appetite is put in abeyance, the food-supply is cut down, and so the viscus comes round again. To encourage a child under these circumstances to eat is cruelty towards it; and still worse is it to tempt it to eat more. This plan only defeats itself. If we saw a weakly child each day induced to carry a weight,—not a great weight, perhaps, but beyond its puny powers,—and each day growing feebler and not stronger, we should all recognize the folly, the cruelty, the wickedness indeed, of persisting in the ill-starred plan. Just so with the incompetent liver. It does not strengthen it, but rather embarrasses it, to feed the child on the food which is suitable enough for the athlete in training, or for the naturally strong man stricken down by an acute illness, where the craving appetite of convalescence tells of digestive power. But the appetite of the delicate child tells of a very different state of affairs in the commissariat department. Magendie made many experiments in relation to uric acid, and Müller comments on them as follows: "These experiments have thrown some light on the causes and mode of treatment of gout and calculous disorders. The subjects of these diseases are generally persons who live well and eat largely of animal food. Most urinary and gravelly deposits, the gouty concretions, and the perspiration of gouty persons, contain an abundance of uric acid,—a substance into which nitrogen enters. By diminishing the proportion of azotized substances in the food, the gouty and gravelly deposits may be prevented." But Magendie, Müller, and others have produced comparatively little effect upon the public mind. Those who have studied the associations of the lithic diathesis, and the causes in action which foster and favor it, denounce the injudicious if well-meant endeavors to give strength to the weakly system by supplying it freely with animal food. Lean meat, raw meat minced, and beef tea are so much poison to such a child. No doubt this outspoken expression of opinion will give offence to many. Then they may just take offence; and I will bear their wrathful outpourings with such composure as comes of a clear conscience.

Not rarely, too, the child craves for tasty dishes, sandwiches with potted meats, and welcomes with gusto the viands pressed on it. But this, again, does not alter the facts. The inexperienced child is surely not the best judge of what is good and desirable for it. The Creator has supplied milk for young creatures, and we may depend upon it we will not err far in following the lead so given us.

Milk contains its albumen in the form of casein, a form which seems to others as well as myself to tax the feeble liver much less than meat-fibrin. And milk ought to be the form of albuminoid *par excellence* for the nursery. As to quantity, Prout wrote, "They should be carefully prevented from *too much* even of bread or of milk."

In discussing the lithic diathesis, the late Prof. Miller, of Edinburgh, writes, "In those cases in which the digestion is obviously weak and imperfect, the food must be regulated as to quantity and quality. Nothing at all approaching a surfeit should ever be indulged in; animal food should be taken sparingly, if at all." And anything approaching a surfeit seriously upsets these children; indeed, they are not likely to perpetrate anything of the kind, if not tempted by the viands and spurred on by approval. As the practice of indulging in animal food increases, it becomes more necessary than ever to protect these delicate children. And the amount of animal food now consumed is much larger than it was a century ago. "So late as 1763 the slaughter of bullocks for the supply of the public markets was a thing wholly unknown even in Glasgow, though the city had then a population of thirty thousand."[1]

Betwixt the growth of towns, involving an impure atmosphere, and the increase in meat-eating, the child of the lithic diathesis is now heavily handicapped.

But there is something worse than meat-eating overhanging it, and that is the resort to peptonized food. The feeble liver is protected by proportionately weak digestive organs. In the attempt to feed up these defective organisms with animal food in large quantities, the digestive organs protect the liver by dissolving only a small portion of the albuminoid elements into the soluble peptones, the rest finding its way out of the alimentary canal without doing any harm. Not so with peptonized albuminoids, however. Already rendered soluble, they penetrate nature's barrier, and find their way without any difficulty into the portal vein, and from thence to the liver. Nature's guard is broken through, and the incompetent liver is flooded with albuminoids; and, further embarrassed by its burden, the liver has to struggle as best it can with this plethora of azotized matters far in excess of the body-needs. Surely, if it were designed further to embarrass the liver and rivet on it the fetters of the lowly uric-acid formation, no plan could afford a better prospect of success than this. It is not likely to rise to the normal urea-formation when thus overweighted. It is very necessary to speak out vigorously on this subject, and to protect the feeble organism from the violence of its would-be friends, who, however, in this matter are, as a matter of fact, its deadly enemies.

To make a child strong by giving it strong food in liberal quantities has a simplicity about it which is very attractive, especially to those who are not familiar with the body-processes.

Light food is desirable. Sandwiches cut thin, and the butter rubbed well into the bread, are not objectionable. Fish of all kinds, especially white-fish and flatfish, are good after the age of three. Before then milk, plain boiled, or in the form of milk-puddings, is desirable. Where the palate, as is not unfrequently the case, revolts against milk in any form, then the next best matters must be adopted. The fish should be served up with genuine melted butter, or, if this is objectionable, a little baked flour may be added, to make it resemble the ordinary melted butter, so called,—

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[1] McCulloch's Statistical Account of the British Empire, vol. ii. p. 502.

an indifferent form of paste, too often. Sweetbreads and other glandular matters are unobjectionable. Small quantities of chicken may be permitted. Where the appetite is very defective and below the body-needs, it may be tempted by a small piece of game, or a small bird.

Fat in all forms is very excellent for these children, but the difficulty lies in getting them to take it. In the visible form of a piece of sweet animal fat it is simply loathed by most of them. To use a phrase in vogue among mothers, it must be "smuggled in." Sandwiches cut thin, and made with slices of cold boiled bacon, are commonly relished and agree well with the strumous. Their potatoes should be baked and the peel removed, well buttered, with some pepper and salt. Or, if boiled, the potato should be mashed and mixed with plenty of butter and some pepper and salt, and be put into a basin and placed in the oven for an hour. All farina that has not been thoroughly exposed to prolonged heat will resist the action of the saliva, and, passing into the stomach unchanged, embarrasses that organ no little in its proper duty. Puddings should be made with biscuits or crackers, and not raw flour. The sago and rice should be well boiled before being made into a pudding. Bread-and-butter puddings are excellent. Oatmeal is good, not only from the amount of fat it contains, but also from the fact that it has made the acquaintance of heat in the miller's drying-kiln.

Why have the crusts of bread been chosen for the "pap" of infants? The "why" is buried in the darkness of the past, but it was a wise choice. The child of lithic diathesis can no more deal with raw starch than it can struggle with a visible lump of fat. Its suet pudding should be made with a mixture of ordinary flour and baked flour in equal parts, and the suet chopped very fine. The liquid fat of fried bacon is most digestible, and the child should be allowed to dip its bread in it, or have it crumbed into the fluid fat. Cream it should have to its stewed fruit. If these matters were more attended to, there would be less necessity for resort to cod-liver oil. Out of five persons taking cod-liver oil, probably only two require fat in that particular form, while three take it because it is the only fat put up in such a form as can readily be purchased. For the three, other forms of fats are preferable; but for the two, cod-liver oil is the only form of fat they can assimilate, and there is no choice for them, however the palate may protest. There is nothing magical about cod-liver oil; it is merely the most digestible form of fat, and therefore is a godsend to many. But it is by no means the best fat. It is even more digestible still when formed into an emulsion. A new competitor is in the field, in the form of condensed cream,—a natural emulsion. This is excellent, as it can be readily procured, is very palatable, and very wholesome. It may not be quite so digestible as cod-liver oil, and so is of little or no avail to some children. But for the much larger proportions it is a boon. It can be added to the nursery bread-and milk, or form a cream to milk-pudding when served, or to stewed fruit, or be made into creams for the table. It is also put up with a certain amount of malt extract, and the admixture is most palatable, except to those who object to the taste of malt in any form. Such preparations will be very acceptable to a large class of persons, and specially town children of the lithic diathesis, and also those who dislike cod-liver oil. In the country, where fresh cream and milk can readily be procured, these preparations are uncalled for. A cup of well-boiled milk allowed to get cold, with the yolk of an egg beaten up in it, and a little sugar, with a pinch of grated nutmeg, is excellent. Or it may be converted into junket if a more solid mouthful is desired.

There are other matters now on the market,—viz., prepared foods. These consist of malt and flour which has been already subjected to heat, so that the starch is largely converted into soluble dextrin. There are various forms of them, valuable, or not, in strict proportion to the care manifested in the preparation. The less sweet go well with a cupful of broth or other meat infusion. The sweeter forms go well with milk. A pint of milk well boiled with a tablespoonful of Mellin's Food is a capital food for a child or adolescent of the uric-acid formation.

All food should be given in small quantities at once, and at repeated intervals. Very often the child is a very small eater; and if attempts are made to induce it to eat more, a keen watch should be kept over the urinary excretion to see if the lithic formation is thereby increased; and if it be so, then the attempts must be moderated.

Alcohol in all its forms is contra-indicated, and should be given only when the necessity for it is very obvious and unmistakable.

The clothing should be light and warm, whether as to day-clothes or night-clothes. The liability of such children to catch cold should ever be borne in mind. The hands should be gloved in cold weather, and the feet should always be well shod. No cheap shoes should be got for these children. They are not good heat-producers, while, on the other hand, they lose heat very readily. It is well to bathe these children in sea-salt and water, which is a tonic to them. If taken to the sea to bathe, the immersion should be brief, and the child should not be taken to the water till two hours after breakfast. The morning dip is well enough for the robust, but not for these children of the lithic diathesis. The same principles must be carried out at mineral springs.

As to medicines, of course the uric-acid solvents, lithia and potassium, come first. The soluble urates of potassium and lithia are more easily got rid of than the comparatively insoluble urates of soda and ammonia. Lugol found potassium very useful with the strumous, giving them relief by getting rid of the uric acid. And this practice has been followed with advantage. Children are usually fond of effervescing drinks, and the effervescent citrates of lithia and potassium are taken readily. *Potus imperialis* sweetened with malt extract is capital. The fluid, too, is good for them, the insoluble uric acid requiring a certain bulk of fluid for its solution, whether it be in child or adult. In strumous children Lugol found iodine highly useful. The syrup of the iodide of iron is frequently indicated. Hæmatics are often required. These children, as a rule, require potash with the chalybeate. Probably this explains why the old *mistura ferri composita* was held in such high repute. And frequently it has fallen to my lot to see the addition of potash to an iron mixture give excellent results. The ammonio-citrate of iron goes well with the bicarbonate of potassium. But children of the lithic diathesis do not bear chalybeates in large quantities at all well. The iron readily upsets their livers. A little arsenic, as Fowler's solution, is often useful. There is also another drug often of great service. Regarded with suspicion in consequence of the gross abuse of it in the early half of this century, every form of mercurial has been abandoned by many practitioners; but its use, as differentiative from its abuse, is coming on again with all thoughtful physicians. Some of our best practitioners in England never gave it up even in the darkest days of its discredit. After enumerating the other medicinal measures, Prout went on, "By the aid of these means, and the occasional employment of mild purgatives or alteratives, as the *hydrarg. cum cretâ*, I have in a great many instances seen the deposition of lithic acid kept in abeyance during the whole period of childhood, and after the age of puberty cease altogether." It is evident, from the literature of that day, that physicians made systematic attempts to help the system to rise above the primitive uric-acid formation; and it seems to me that this practice might be revived with advantage. How it dropped out of sight in recent years, it is not easy to say. The testing of urine for albumen and sugar has thrust urinary sediments into the background. If these two were absent, all was right. A more shallow doctrine was never preached. The significance of deposits visible to the naked eye will not be very long in regaining its old position. Whenever the urine of a child presents lithic deposits as a matter of habit, the sooner that child is carried to a thoughtful physician the better for all concerned.

The subject can easily be summed up. The newly-born child possesses the uric-acid formation as a normal matter. But it gradually outgrows or rises above this lowly formation, and leaves it behind at puberty,—*i.e.*, if it is equal to the urea-formation. A delicate child fails to achieve this. And then it becomes our duty to give it the requisite help, if we possibly can, by the application of the principles just laid down.

DIABETES MELLITUS.

By GEO. B. FOWLER, M.D.

Although diabetes mellitus is a comparatively rare disease at any period of life, it does occur in varying frequency from birth to old age. The highest point of liability to this affection is between forty-five and fifty years, whence the line slopes down precipitately to the two extremes of life.

So rare is diabetes in infancy and childhood that few of the text-books devoted to pediatrics mention it at all, and, as a rule, general treatises on the subject usually fail to speak of any peculiarities which it presents when occurring in young subjects.

Sir William Roberts gives a table, prepared from the Registrar-General's Report for 1851–60, for England and Wales, showing the number of deaths, the age, and the relative frequency as to sex :

Period of Life.	Under 5 Years.	5–10 Years.	10–15 Years.	15–25 Years.	25–35 Years.	35–45 Years.	45–55 Years.	55–65 Years.	65–75 Years.	75 and up.	Total.
Deaths in males	28	40	97	378	468	502	550	500	364	105	3032
Deaths in females	23	42	78	220	282	261	247	191	144	26	1514
Total males and females	51	82	175	598	750	763	797	691	508	131	4546

From the same report we gather the following general facts: that, whereas from 1851 to 1860 the total number of deaths from diabetes was four thousand five hundred and forty-six, from 1871 to 1880 there were nine thousand three hundred and three.

The table given by Dr. Dawson Williams[1] shows at a glance several very interesting facts: that diabetes, and urinary diseases in general, are yearly becoming more frequent, and that they are faster on the increase than are nervous affections.

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[1] Pathological Soc. Trans., 1883.

MEAN ANNUAL RATE OF MORTALITY IN ENGLAND.

Annual Deaths to 1,000,000 *Living.*

YEARS	1850 to 1854.	1855 to 1859.	1860 to 1864.	1865 to 1869.	1870 to 1874.	1875 to 1879.	Average, 1850 to 1879.
Deaths from all causes	22,299	22,052	22,248	22,760	22,019	21,250	22,105
Deaths from nervous diseases	2,777	2,758	2,823	2,859	2,817	2,812	2,808
Deaths from urinary diseases	190.6	227.	270.6	320.2	352.2	420.	296.9
Deaths from diabetes	23.	24.8	28.4	32.2	35.2	40.6	30.7
Deaths from gout	12.4	13.2	13.4	18.2	20.8	25.6	17.3

The proportion of males to females varies distinctly with the age, as will be seen in the first table above. It is about equal up to ten years: after that to the end the male is vastly more liable. Through the courtesy of Dr. John T. Nagle, Register of Vital Statistics of New York City, we are enabled to present this table, showing

THE NUMBER OF DEATHS FROM DIABETES MELLITUS IN THE CITY OF NEW YORK FOR TEN YEARS,

Population estimated at 1,400,000 *as an Average.*

YEAR.	UNDER 5 YEARS.	5–20 YEARS.	OVER 20.	TOTAL.
1878	...	2	40	42
1879	...	2	35	37
1880	1	3	40	44
1881	...	...	43	43
1882	2	1	43	46
1883	...	4	64	68
1884	1	2	65	68
1885	...	2	61	63
1886	...	9	73	82
1887	...	2	103	105

The rarity of the affection at the early periods of life is clearly shown by such statistics, and we need not be surprised at the non-mention of it by most standard authors. Prout, out of seven hundred cases, saw only one in a child of five, and about a dozen between eight and twenty, four of whom were females (quoted by Day). Roberts, with his vast experience, saw one case in a boy of three years. West had seen only one case at three and a half years.[1] A case is reported by Dr. Thompson[2] of a boy of five years. During the past year we saw reported a case as occurring in a child of twenty-one months, and within a short time the writer had under his care a typical example of the disease in the person of a girl of four years.

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[1] Diseases of Infancy and Childhood, Philadelphia, 1860, p. 534.

[2] Glasgow Med. Jour., New Series, 1885, xxiii. 59.

Etiology.—The cause of diabetes in children is as obscure as it is in adults. Cold, shock, traumatism, diet, heredity, locality, hygienic surroundings, and many other influences have all been accused. After carefully studying the few reported cases, and our own case, we are inclined to consider heredity, and especially a phthisical or scrofulous history, as decidedly predominant as an etiological factor, so far as the development of this disease in children is concerned.

Pathology.—It may be safely asserted that no constant lesion has been found which distinguishes diabetes mellitus. The results of post-mortem examinations are as various as the theories regarding the cause of this disease. Proceeding from the head down, every organ and tissue at times has been found either normal or altered. The familiar glycogenic function of the liver caused eager search to be directed to this organ for a solution of the problem. Hundreds of ingenious experiments have been performed, but with no fixed result. With Bernard's marvellous discovery of the seat of the vaso-motor centre in the fourth ventricle, and the effect of its destruction or irritation upon the liver and urine (temporary glycosuria), an enormous amount of work has been done by way of investigation into the nervous influences possible to be brought to bear, and capable of imitating or establishing this disease. Every comprehensive work on physiology gives a list, and describes at length the most philosophical of these experiments and the most plausible results; and such knowledge, being our stock in trade, should not be here detailed. A great many of the lesions found after death have nothing to do with the etiology of diabetes. They are the degenerations consequent upon the disease.

Bearing in mind the well-established nervous endowments of the liver, let us look for a moment at the alimentary canal. To a casual observer, as well as to one versed in physiology, the appearance of sugar in the urine seems a decided upset of the natural order of things. In this connection we will quote our own words from a recent article upon a kindred subject:[1]

"The materials introduced into the body for its maintenance are the albuminoids, carbo-hydrates, hydrocarbons, salts, water, and oxygen. The substances which under physiological conditions appear in the excretions are, practically speaking, urea, salts, carbonic acid, and water. The albuminous matters serve their purpose, and, being non-diffusible, are converted into the crystalloid urea, in order to be gotten rid of. The carbo-hydrates and hydrocarbons, having a great affinity for oxygen, are by this agent converted into water and carbonic acid; while the remaining proximate principles, the mineral salts and water, being very diffusible and little prone to change, are excreted under their own forms. Thus, theoretically, at the start it appears that, under normal conditions, the urine should contain none of the organic ingredients of the food, nor any of their immediate derivatives (albumen, peptone, paraglobulin, metalbumen, glucose, maltose, chyle)."

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[1] The Significance and Detection of Traces of Sugar in Urine, Dietetic Gazette, October, 1888.

When such a thing does occur, then, we naturally direct our inquiry to the digestive processes. Sure enough, experimental and pathological interferences here are known to produce glycosuria, chief among which are the ingestion of excessive quantities of saccharine or starchy food, the production and absorption of poisonous by-products of digestion, thereby irritating the liver and preventing the proper performance of one of its functions, and the pressure of tumors, whether new growths, fæces, or what not, upon the cœliac plexus, thus modifying the blood-supply to the liver and digestive glands; and, finally, it has been established that the pancreas is diseased in about one-half the autopsies made upon persons dead of diabetes. Even the exact pathological condition of this organ under these circumstances is not constant. It may be atrophied, or degenerated, or cancerous, or may contain calculi; and sometimes the gland has almost entirely disappeared. This frequent connection of a diseased pancreas is certainly very striking. But it is difficult to explain the diabetes by the arrest of the function of this organ. It has been suggested that it operates simply by pressure upon the sympathetic plexus beneath. What causes the disease in the other half, where the pancreas is found intact? These cases have been attributed to nervous influences, worry, exhaustion, emotion, etc., and to those causes already spoken of as heredity, traumatism, cold.

Ebstein, in his recent work on the subject, offers an ingenious theory, based upon experience and exact experiments, that diabetes is due to an inherent defect in the protoplasm, whereby too little carbonic acid is liberated. He holds that he has proved that carbonic acid has an inhibiting effect upon the diastatic ferments, and that when this gas is present in too little amount the various diastatic ferments throughout the tissues act too vigorously upon the omnipresent glycogen and throw an enormous excess of sugar into the circulation. This explanation ignores any local cause. It very well includes all causes in a general systemic fault, which, it seems to us, must itself be due to some chief determining local derangement to begin with.

Pavy, as is well known, believes that the whole trouble is due to imperfectly dearterialized venous blood, due to vaso-motor paralysis, especially of the vessels of the chylopoietic system. Seegen has recently published an account of his latest experiments, which lead him to assert that the liver makes sugar out of albumen and fat and makes glycogen out of food carbo-hydrates.

In a recent communication to the French Academy of Medicine Lancereaux expresses the most plausible opinion when he says that diabetes does not mean a fixed and single pathological condition; that it includes several processes, in one of which the pancreas is affected. Another form is characterized by an increase in flesh, chronic joint-disease, and not usually associated with pancreatic alterations. This is generally called constitutional or fatty diabetes. Then there is a third variety, due to affections of the nervous system, traumatic or emotional. The symptoms here are mild, there are no pathological organic changes, and recovery is the rule.

This brief outline will serve to show how diverse are the pathological views, and how difficult it is to unravel the mystery of this fatal affection.

Symptoms.—These vary but little in children from those common to adults. There is the same great thirst, emaciation, profuse urination, excessive appetite. A peculiarity presented in children is the rapidly fatal course. These points can be best presented by rehearsing a couple of histories. The first is that of Dr. Thompson, from the *Glasgow Medical Journal:*

A. H., aged five years, was brought to my house on the 9th of August, when the mother gave the history as follows. The patient enjoyed excellent health until three weeks ago, when he was noticed to drink from the water-tap every few minutes. His appetite, also, was extraordinary. At first these were attributed to a childish freak, and he was forbidden to take so much water. When water was withheld he satisfied his craving for fluid by drinking castor-oil, which he stole for the purpose. The quantity of urine passed daily was large. Bowels had been all along confined. Since his illness was first noticed he has been getting daily more and more easily tired, paler, and thinner.

When seen by me the patient was pale and thin, the muscles wasted, soft, and flabby, and he weighed with his clothes thirty-two pounds and fourteen ounces. Thirst was severe, and while being examined he had to be supplied with water, which he drank greedily. Immediately after, he passed a quantity of pale acid urine having a sweet taste and a specific gravity of 1035, but free from albumen. Fehling's solution and fermentation demonstrated the presence of a large percentage of grape-sugar. The thoracic organs seemed normal. The abdomen was tense and rather hard, but neither pain nor tenderness was complained of. Liver natural, and no evidence of abdominal fluid. Temperature 99° F.

The child died after seven days, the specific gravity varying between 1036 and 1034 and the daily quantity being from five to ten pints. Dietetic treatment is alone mentioned.

The second is the example which lately came under the writer's care, and which had the following history:

L., aged 4 years. Eight months before, the mother had observed the child to be "out of sorts,' and soon afterwards to develop great thirst and very frequent urination. A homœopathic physician diagnosed Bright's disease, but apparently discovered no "similia," for the child grew gradually worse, and when I saw her she was very weak, pale, and thin, and disposed to sleep most of the time. During the first consultation the little one begged for water several times, and asked to be allowed to urinate. These incidents immediately aroused the proper suspicion, and on further examination the urine was found to have a specific gravity of 1040, to contain seven grains per ounce of sugar, and the daily quantity to be three and one-half pints.

Careful inquiry failed to elicit any history of exposure to cold or wet, and neither traumatic nor emotional cause could be discovered. The child had previously been very well. Upon interrogating the parents, I found them both to be decidedly below par as regarded both physical and mental development, and their general appearance was of the tallow-candle sort. Each was poorly nourished, and had thick coarse hair, shiny skin, and glistening deep-set eyes. As a pair they exhibited a degree of bodily and mental sluggishness rarely encountered. A grandparent on either side had died of phthisis.

The child was put upon a very restricted diet (diabetic) and five drops of the fluid extract of ergot three times a day. Within three days the quantity of urine was reduced to two pints, and all the sugar had disappeared. The thirst was also markedly relieved. Disposition to sleep became more and more pronounced, however, and at times the breathing was very labored. These symptoms convinced the parents that my remedies were too strong, and they again called in a homœopathic physician. I learn that death ensued twenty-four hours afterwards.

Diagnosis.—Of all diseases, there should be no difficulty in discovering diabetes mellitus. The emaciation, thirst, excessive urination, and saccharine urine form a combination of signs which distinguish this affection and no other. Of course, should a case be encountered in its incipiency, especial care must be exercised in the detection of sugar in the urine. When this substance is constantly found, in however small amount, together with any or all of the above symptoms, the diagnosis is made.

Prognosis.—No recoveries from diabetus mellitus occurring in children have been reported. In fact, the disease is here very rapidly fatal.

Treatment.—The treatment differs in no respect from the course pursued with adults. Our own success with a certain number of cases in older people led us to try ergot in the one narrated above. The very favorable effect upon the urinary symptoms was in harmony with our former experience. Whether an earlier administration of this drug, or some other plan of treatment, would have been of permanent benefit, it is impossible to say.

PART III.

DISEASES OF THE RESPIRATORY TRACT.

NASAL OBSTRUCTION.

By JOHN NOLAND MACKENZIE, M.D.

We are told in Genesis that, when God made man, it was not into his mouth, but into his *nostrils*, that he breathed the breath of life. The disastrous consequences to the organs of respiration, audition, and voice-production from occlusion of their natural atmospheric channels are too often lost sight of by those who, unmindful of this truth of scriptural physiology, sum up the varied functions of the nasal apparatus in the terse proposition, The nose is the organ of smell.

The influence of nasal obstruction in the causation not only of morbid conditions of the whole respiratory tract and middle ear, but also of pathological changes in other and remote organs of the body, is no longer a matter of interesting speculation, but is grounded on the firm foundation of every-day clinical fact and experience. The removal of nasal obstruction in young children is of especial importance, for in them it means interference with the act of suckling and consequently with the maintenance of life.

Obstruction of the nasal fossæ may be acute or chronic. In the following pages only the subject of permanent or chronic obstruction will be discussed.

Etiology.—The lumen of the nasal passages, or that portion which is included between the septum on one side and the turbinated bodies on the other, varies greatly in capacity within the limits of perfect health. It may be congenitally narrow enough to interfere seriously with respiration, and it was this congenital anomaly, doubtless, of which Sylvaticus wrote over two centuries ago.[1] The present article deals exclusively with those malformations which predispose to or cause obstruction of the nasal passages. Anomalies of this kind may be congenital or acquired, and may be separated into those which affect the posterior, those which affect the middle, and those which affect the anterior third of the nasal fossa.

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[1] Consiliorum et Responsorum medic. Centuriæ IV., Patav., 1650, cent. II. consil. 24.

The posterior third of the nasal passages may be more or less completely obstructed by anomalous conditions of the nasal pharynx or by anatomical peculiarities of the posterior nares.

The nasal passages are much more frequently than the pharynx the seat of congenital abnormalities,—a fact probably explicable by the comparative structural simplicity of the latter as compared with the more complicated architecture of the former.

That malformations of the naso-pharynx are of rare occurrence is the inference which follows from their cursory mention in works on teratology, and the infrequency with which isolated cases are encountered in periodical medical literature. If we consider, however, the complex process involved in the embryological evolution of this region, if we reflect that many of its deformities, indirectly removed from sight, may be compatible with the perfect comfort of their possessor, and therefore come only accidentally under medical observation, and if we bear in mind the notable infrequency with which the naso-pharyngeal cavity is examined after death, it is quite possible that departures from its normal structure may be more common than is generally supposed.

In that wonderful book[1] of which it has been said that it is as full of variety as nature herself, Pliny the Elder tells us that children born in the seventh month frequently have the ear and nose imperforate. Whether the observation of the great natural historian be correct or not, it is quite certain that occlusion of the posterior nares is the most common of congenital naso-pharyngeal anomalies. The occlusion may affect one or both nostrils;[2] may be membranous or bony.[3] The orifices of the posterior nares may be alone implicated, or the nasal fossæ may be obliterated in their entirety.[4] Obliteration of the choanæ occurs when the nose is absent, as in cyclopean monsters,[5] or rudimentary;[6] or it may constitute the sole aberration from the normal in the individual. This malformation, when congenital, seems to be incompatible with the independent life of the fœtus.[1] Fusion of the choanæ into one is occasionally observed associated with absence of the vomer.[2] The position of the posterior edge of the vomer is, according to most anatomists, always median and perpendicular, deflection in this situation being probably exceedingly rare.[3]

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[1] Nat. Hist., lib. xi. cap. 59.

[2] Vicq d'Azyr, Mém. de la Soc. de Méd., 1776, p. 315; Otto, Handbuch d. path. Anat., Breslau, 1814, S. 201; Roederer (quoted by Meckel, Handbuch d. path. Anat., Leipzig, 1812, i. 406); Plancus, Mém. de Berlin, 1761, p. 73; Otto, op. cit.; Cohen, Diseases of the Throat, etc., 1879, p. 305; Luschka, Der Schlundkopf des Menschen, Tübingen, 1868, S. 27; Fränkel, Ziemssen's Cyclop., Amer. ed., vol. iv. p. 11; Bitot (Knight); Emmart (quoted by Luschka); Voltolini, Die Anwendung d. Galvanocaust., Wien, 1870, pp. 243–262; *et al.*

[3] Oberteufer, in Stark's Neuem Archiv, ii., St. iv., S. 640; Otto, loc. cit.; Littré, Mém. de l'Acad. des Sci., 1701, p. 120. Bony occlusion may result from the separation of the nose and pharynx by means of a bony wall or plate, or may be produced by the fusion of the superior maxilla, palate, and ethmoid. On bony occlusion of the posterior nares see an interesting article by Knight, of New York (Medical News, Phila., Nov. 10, 1888), and Hubbell, of Buffalo (Trans. State Med. Soc. of New York, 1886).

[4] Rohowsky, De Choanarum Obliteratione. (Meckel, Otto.)

[5] Vrolik, Soemmering (Paget, Todd's Cyclop., art. "Nose"), Borrechius, Plouquet (Otto, loc. cit.).

[6] Maigrot (Roux, Jour. de Méd., t. xv. p. 142, Meckel). Roloff (De Monstris, Venet., 1749, Meckel)

Very rarely it is divided vertically into two halves, as in the cases recorded by Lefferts[4] and Schrötter,[5] and Harrison Allen[6] refers to a specimen in the Wistar and Horner Museum in Philadelphia, where the vomer was strengthened on each side by a delicate bony process from the palate bones. Finally, the capacity of the naso-pharynx varies greatly in different individuals, especially in its antero-posterior diameter, which is often notably diminished, and Lennox Browne[7] asserts that occlusion of the nostrils may result from angular curvature forward of the upper cervical vertebra. I have seen several cases of marked lateral deviation of the posterior wall.

Obstruction of the anterior segment of the bony nostril is not infrequently caused by departures from the normal structure of the vomer, the turbinated bones, and the ethmoid. These anomalies vary greatly in character and degree, and it is sometimes difficult to say where malformation ceases and hypertrophy begins. As the result of excessive development the position which the turbinated bones assume is often a striking one. Well-marked hypertrophy of the upper bone is comparatively rare,—of the inferior, much more common: in the latter case the bone presents the appearance of an exostosis from the inferior nasal meatus. The most interesting departures from the normal position occur, however, in the middle turbinate. This bone may grow directly inward, displacing the septum, or downward, reaching the lower meatus, or it may grow directly inward and then suddenly pursue a downward and outward course. Occasionally in the rhinoscopic image it has an appearance as if curled upon itself like a snail. One of the most striking forms which the middle turbinated bone occasionally assumes is the abnormality first described by Santorini,—the conversion of its anterior end into a large, hollow, bony bladder-like body, which to the uninitiated may readily appear as an exostosis or even a polypus.

Obstruction of the middle third of the nostril may be caused also by deflection or dislocation of the vomer or the perpendicular plate of the ethmoid. In the former case it generally takes place at the junction of the former with the cartilaginous septum, and the condition of the latter is, according to Harrison Allen,[1] one of hyperostosis of the sutural line.

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[1] Ronaldson, Edinburgh Medical Journal, May, 1881, p. 1035.

[2] Roloff (loc. cit.). Roederer (loc. cit.). Fernet (Bull. de la Soc. Anat., 1864, p. 130). Blandin, Anat. topograph., p. 74.

[3] Welcker, however, found asymmetry of the choanæ thirteen times in thirty-seven cases. (Ziem. Monatsschrift f. Ohrenheilkunde, February, 1883, p. 23.)

[4] Phila. Med. News, January 7, 1882, p. 12.

[5] Laryngologische Mittheilungen, Wien, 1875 (Lefferts, loc. cit.).

[6] Amer. Jour. Med. Sci., January, 1880, p. 72.

[7] Brit. Med. Jour., 1878, vol. ii. p. 282.

Not infrequently it is inclined neither to one side nor to the other, but presents, usually in its bony portion, but sometimes at the junction of the cartilage with the vomer and ethmoid, an oblique, rounded, bony ridge, which produces more or less occlusion of the nostril, into which it projects. The opposite surface of the septum corresponding to that of the anomaly is usually concave. This form of septum, which has been carefully studied by Zuckerkandl[2] (who found it one hundred and seven times out of three hundred and seventy skulls), did not escape the acute observation of Morgagni,[3] who was the first to describe it.

A very common point of irregularity in the vomer is along its inferior edge, in the neighborhood of the nasal spine, where it is associated with a similar projection of the cartilage, the two together forming a more or less wedge-shaped process, whose apex lies across the floor of the lower meatus. Loewenberg,[4] who has made a number of sections through the vomer and cartilage at this point, calls attention to the fact that the spurs which are commonly seen along the lower edge of the vomer anteriorly at its junction with the cartilage proceed from the bony and cartilaginous parts not being in the same vertical plane, but joining at a dihedral angle, projecting towards one side. The projection is here formed on the one hand by the lip of the vomer, and on the other by the insertion of the cartilaginous portion.

Occasionally an S-shaped incurvation, from above downward, of the bony septum is seen, in which both the vomer and the perpendicular plate are concerned. The posterior edge of the vomer is very rarely deflected. A remarkable case of this kind, where the naso-pharynx was divided into two lateral halves, is recorded by me in the *Archives of Laryngology*.[5]

Now and then obstruction may occur from abnormal growth of the ethmoid itself, so that the bulla ethmoidalis may project into the middle meatus, occluding that channel, or may pursue an inward course, displacing the septum (Zuckerkandl).

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[1] Amer. Jour. Med. Sci., January, 1880.

[2] Normale u. path. Anatomie der Nasenhöhle, etc., p. 48, Wien, 1882.

[3] De Sedibus et Causis Morborum, I., xiv. 16.

[4] Archives of Otology, March, 1883.

[5] July, 1883. Since then three cases of this deformity have been observed. As the result of an examination of a number of crania suggested by the above anomaly, I would call attention to the varying degrees of obliquity which the plane of the posterior nares bears to the horizontal: in some instances the angle is so small that they look almost directly downward. This inclination of the choanæ involves a corresponding obliquity in the posterior edge of the vomer, and coincides with an abnormal inclination of the pterygoid processes and the body of the sphenoid bone. Herein, too, lies the anatomical explanation of the variations in the angle which it is often necessary to give to the rhinoscopic mirror before the image of the nares appears in the glass. In the production of the above malformation three factors are doubtless concerned: (1) obliquity downward and backward of the body of the sphenoid bone and basilar process of the occipital; (2) abnormal curvature backward of the vomer, associated with marked obliquity of the posterior orifices of the nasal fossæ; and, possibly, (3) an unusual height of the bony palate.

Congenital obstruction of the anterior third of the nasal fossa is very uncommon in children otherwise well formed. Especially is this true of bony occlusion.[1] A remarkable case is recorded by Littré in which the mouth and nasal passages were closed by a membrane which was continuous with the neighboring skin. On the other hand, the anterior nares are not infrequently closed by a number of pathological processes, notably syphilis and lupus. Several cases have been reported in which obstruction in the anterior nasal chamber was produced by the upward growth and projection of the eye-teeth.

Growths of the nasal passages are uncommon in young children. Especially is this true of the ordinary gelatinous polypus so frequently met with in the adult. Morell Mackenzie[2] has never seen the affection under the age of sixteen, but quotes a case from Mason[3] in which nasal polypi were removed from a boy of twelve. I have removed from two children (brother and sister), aged four and five respectively, mucous polypi the size of Blue Point oysters. In the one case the growth became visible during the first year of life, and in the other there were many reasons to believe in the congenital origin of the neoplasm.

Very extensive hypertrophy of the turbinated tissues and other portions of the nasal chamber are also comparatively rare in very early life, but it not infrequently happens that, from some vice of constitution, the intranasal tissues undergo hypertrophic changes which are fugitive and which go on rapidly to atrophy. Obstruction of one or both nostrils from dislocation or malposition of the nasal septum is, on the other hand, a common accident of childhood, whilst the nasal passages rank with the external auditory meatus as convenient receptacles for the buttons and other foreign bodies which children delight to introduce into these cavities.

The walls of the nasal fossæ may be connected by synechiæ, which may be membranous or bony, congenital or acquired, either from traumatic influences or through adhesion from pathological processes. Synechiæ are most commonly found between the turbinated bodies and the septum, but may occur in other portions of the fossæ. Sometimes the nasal pharynx and nasal passages may thus be completely obliterated, notably in the case of syphilitic ulceration. Obstruction of the nostril may also occur from syphilitic fibroid degeneration of the nasal structures.

There is one form of nasal obstruction, finally, to which we may give the paradoxical title of *obstruction of patency*, and which consists in an extreme degree of capacity of the nasal chambers. It is a familiar clinical fact that, other things being equal, detention and decomposition of the secretion are more likely to occur in an abnormally dilated nostril than in one in which the normal anatomical relations of the structures are preserved. This is readily explicable by the altered physical conditions which abnormal widening of the nasal cavities involves. The greater the calibre of the passage, the feebler the expiratory current of air, and the more difficult, therefore, the voluntary removal of secretion. Imperfect ventilation and stagnation of the air in the nasal chambers follow, too, diminution in the force and rapidity of the inspiratory stream; the cold, dry air, laden with impurities, diffuses itself in the spacious compartment, in contact with a membrane whose functions are often suspended by disease, and which, accordingly, is incapable of fulfilling its physiological destiny in the processes of normal respiration. Thus to retained and decomposing secretion is added an unfiltered, vitiated atmosphere, and conditions are established which favor putrescence and the consequent development of ozæna.

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[1] Potter, Buffalo Med. and Surg. Jour., September, 1888; Jarvis, Trans. Amer. Laryngological Assoc., 1887.

[2] Diseases of the Throat and Nose, vol. ii. p. 356, London, 1884.

[3] Med. Soc. Proc., London, 1872–74, vol. i. p. 156.

Effects of Nasal Obstruction.—The evil effects of nasal obstruction may be felt in almost every organ of the body. So important is a proper discharge of the nasal functions, not only to the structures directly involved, but also to the general welfare of the individual, that the abrogation or suspension of the vital properties of the intra-nasal tissues may be looked upon as one of the most serious obstacles to the enjoyment of normal physiological life. This is especially true in early childhood, when growth and development are going on with rapidity, and when the demand for healthy respiration is accordingly all the more imperative. It is a remarkable fact that congenital occlusion of the nares seems to be incompatible with the viability of the fœtus; whilst the bad health and stunted growth of children suffering from nasal obstruction are matters of every-day occurrence, unfortunately too frequently overlooked.

The immediate effect of the removal of a nasal or post-nasal obstructive lesion, especially in children, is often marvellous. From Liliputian dimensions they reach with almost magic rapidity the full measure of their normal growth.

Although the complications to which nasal obstruction gives rise were partially recognized by some of the earlier writers, it is only within a comparatively recent period that its pathological importance has been fully appreciated, and that only by a few whose special studies have led them to the full recognition of its sequels. Many an aural catarrh has been allowed to end in hopeless deafness, many a naso-laryngeal inflammation has become inveterate and incurable, from failure to recognize the evils which result therefrom; and were the statistics of such cases carefully compiled, they would appear to many in the form of a revelation. So important is their relationship that it is my invariable rule to begin the examination of the throat and ear with an exploration of the nasal fossæ and retro-nasal space.

When the symptoms of this condition are more closely examined, it becomes evident that obstruction of the nostrils is equivalent to interruption of their functions as organs of respiration, olfaction, audition, and voice-modification, and the phenomena to which it gives rise will therefore depend upon the anatomical seat of the obstruction and upon the physiological properties of the structures that assist in its production.

Obliteration of the upper meatuses or malformation of the nasal roof is accompanied by interference with the sense of olfaction and by the varied train of morbid impressions which follow its perversion. Over a century ago Morgagni called attention to deflection of the septum as a not infrequent cause of unequal distribution of the nervous power of smell, and related a case in which, from obliquity of the crista galli and consequent diminution in number of the olfactory foramina, this sense was supposed to have been less acute in the nostril corresponding to the narrower side of the cribriform plate. Just here let me observe that the ability to respire with freedom through the nose by no means negatives the existence of obstruction: the upper nasal chambers may be crowded with growths and the pharyngeal vault covered with vegetations, and yet respiration go on with perfect ease. This is a fact which is overlooked by many practitioners of medicine, and the absence of obstruction is too often inferred from the passage of air through the nostrils when the mouth is closed.

Should the obstruction occur in the inferior meatus, the mouth becomes the channel through which the air passes to and from the lungs, and nasal gives place to buccal respiration. Thus the air reaches the delicate lining membrane of the lower respiratory tract in a state unfit for respiration. Hence arise hyperæmia and chronic inflammation of the pharyngo-bronchial membrane, with their associated alterations in the voice.

Nasal obstruction in children is the fertile source of many incurable respiratory and aural affections in after-life.

In nasal obstruction of long standing, chronic inflammatory changes are sometimes induced in the bronchial and pulmonary mucous membrane, which are exceedingly difficult to deal with even after the original cause is removed, and this has doubtless given rise to the popular idea that "catarrh" is the forerunner of consumption. Certain it is that nasal obstruction predisposes, other things being equal, to inflammatory conditions of the respiratory tract, and that the practical physician cannot afford to overlook the influence which it exerts in their production. In this country the vast majority of the cases of chronic laryngitis originate primarily in disease of the nose, and many a winter cough is allowed to go on from bad to worse because of failure to recognize this relationship. I am furthermore convinced that nasal obstruction may and does awaken diseased states of the lungs, and *in an individual so predisposed may favor* the development of pulmonary consumption. Fränkel states that emphysema frequently coexists with nasal stenosis, and Kussmaul believes that acute hyperæmia of the lung may be produced by the forced inspiration of the air. The vesicular murmur is weakened, feeble, and shortened in inspiration, and only approaches the normal when deep inspiratory efforts are made. Frequently mucous and subcrepitant râles can be heard in different portions of the chest. Attention has been called to certain deformities of the chest-walls consisting chiefly in malposition of the bones and loss of power in the muscular covering. I believe these deformities to be rare, except in very young children.

The influence of nasal obstruction in such cases is of course purely accidental and mechanical. Although the theory of the direct pathological relationship between simple nasal disease and tuberculosis of the lungs has been gravely advanced by more than one observer, it seems scarcely necessary to remark that we must accept observations of this kind with the utmost caution, and require more abundant proof of the alleged facts than those already in our possession.

Besides the part which the nose plays in the processes of olfaction, respiration, and voice-production, it also serves as the channel of conduction of atmospheric air to the middle ear. The aural pressure is kept in a state of stable equilibrium by the constant supply of air to the cavity of the drum through the Eustachian tube. In the natural state this ventilation of the tympanum is continually taking place, not only as the result of the partial vacuum created in the naso-pharynx during the act of deglutition, but also during normal nasal respiration. It follows, therefore, that anything which tends to obstruct the passage of air through the nose will interfere, to an extent varying with the amount of obstruction, with normal aural ventilation, and consequently with physiological intra-tympanic pressure. This diminution of pressure within the cavity of the drum, which can readily be demonstrated experimentally, leads necessarily to inward collapse of the membrana tympani, with consequent abrogation of function in the osseous and muscular apparatus of the middle ear. Catarrhal otitis media, with its long train of phenomena, is the inevitable result; fluid not infrequently accumulates in the tympanum, which finds an exit ultimately by perforation of the membrane and leads to chronic otorrhœa. This same chain of events follows the obstruction of the Eustachian tubes by growths in the pharynx or the pressure of the hypertrophied nasal turbinated structures, or by inflammatory engorgement of the orifices of the tubes themselves. This cuts off the air-supply from the tympanum not only by direct occlusion of its natural channel, but also by interfering with the motions of the velum, and therefore with the opening of the tube by the tensor palati or dilator of the tube. The intimate and direct connection of the blood-supply of the tube and pharynx with that of the middle ear, and their anatomical continuity of tissue, favor, furthermore, the extension of the inflammatory process from the one to the other. Indeed, in very many cases the aural inflammation is merely a symptom of nasal catarrh, and gradually disappears without special treatment upon the removal of its primary cause.

Inflammation of the tube may result in stricture; and in long-standing cases of salpingitis, fatty degeneration of the tubal muscles occurs, with the consequences described above.

These are by far the most common causes of chronic catarrhal inflammation of the middle ear. There is still another way, however, in which morbid conditions of the nose may react upon the circulation and nutrition of the aural chambers,—viz., through the reflex agency of the vaso-motor and trophic nerves.

I have repeatedly called attention[1] to this reflex agency of the vaso-motor and trophic nerves in the production of middle-ear disease, to the recognition of which I was led by the accidental production of symptoms referable to the ear (such as tinnitus, pain, stoppage, etc.) during operative procedures in the nose. Although my experiments upon this point have as yet taken no definite form, it is quite possible that the aural affection in these cases may find its explanation in pathological conditions of the reflex sensitive area which I have shown to exist in the nasal mucous membrane.[2] At least, in several cases I have succeeded in reproducing them by artificial stimulation of this area. This is a fact of considerable practical importance in the solution of many obscure and intractable cases of middle-ear disease whose etiology has been heretofore unrecognized.

It is impossible to exaggerate the part which diseases of the nose play in the production of inflammatory conditions of the middle ear. Between sixty and seventy-five per cent. of all cases of ear-disease originate primarily in morbid states of the naso-pharynx, and the successful treatment of middle-ear catarrh will in the vast majority of instances depend upon their recognition and removal.

The inflammation of the conjunctivæ which is so often observed in connection with nasal obstruction is generally explained by the extension of the inflammatory process through, or occlusion of, the nasal duct; but I am inclined to regard it in many instances as a reflex vaso-motor phenomenon, the vessel-dilatation being kept up by the constant irritation of the sensitive nasal area. In like manner I would explain the recurrent herpes and keratitis which have been observed in connection with this disease, the phenomena in these cases being called forth by trophic disturbances.

The most common result of obstruction of the nasal passages is inflammation of the nasal pharynx. Extension of the inflammatory process into the ethmoid or even sphenoid cells is also met with, and is often a most difficult sequel to deal with. Obstruction of the nasal duct and dacrocystitis are occasional complications; but both these affections and the extension of inflammation to the frontal sinus are comparatively infrequently met with, although popular belief would seem to indicate that catarrh of the latter cavity is the prolific source of all the headache of catarrhal rhinitis.

Nasal obstruction may even lead, in very young subjects, to asymmetrical conditions or imperfect development of the nasal and accessory chambers, and of other portions of the skull on the side corresponding to the seat of obstruction. Indeed, Ziem has shown experimentally that in certain cases nasal obstruction may be an important factor in the production of asymmetrical conditions of the cranium.

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[1] Trans. Med.-Chir. Fac. of Maryland, 1883, and Trans. Amer. Laryngol. Assoc., 1883.

[2] Amer. Jour. Med. Sci., July, 1883

There is one symptom of nasal obstruction to which especial importance must be attached, and for which alone the physician is often consulted. *Dyspnœa on exertion* is one of the most annoying features of the case. Such patients complain that in talking they must frequently pause for breath; that in going up-stairs, walking rapidly, or running,—in fine, in all bodily operations which require unusual exertion,—they get very readily out of breath. Difficult breathing is also present when the mouth is occupied or closed, as in swallowing, smoking, etc. They are accordingly constantly haunted by the dread of heart-disease and consumption. Physical examination, however, fails to detect any cause for the dyspnœa. At times the breathing is perfectly normal and vesicular; at other times a few small mucous râles may be heard posteriorly in the inferior lobes, or in the infrascapular and mammary regions.

Hemorrhage from the nose is a not uncommon symptom of nasal obstruction. It may be small in amount, or may be copious enough to produce considerable depression, and even collapse. It is usually excited by picking, scratching, rubbing, or blowing the nose, by sneezing and coughing, by the separation of crusts, and by a multitude of other exciting causes that determine an increased flow of blood to the nasal membrane. Sometimes such hemorrhages occur at night, from unconscious irritation of the nose with the finger during sleep. Quite extensive loss of blood occurs, however, without the intervention of traumatic influences in chronic inflammatory conditions of the nasal tissues, perhaps from congestion of the cavernous structure and loss of resistance in the erectile cellular walls, or from stoppage of the nostrils, for when the nose is freed of mucus and crusts, or when the redundant tissue is removed and the normal aerial pathway is restored, the recurring hemorrhages cease. The bleeding in many such cases comes from the cavernous tissue. Indeed, nasal obstruction from any cause (deflection of the septum, hypertrophic enlargements of the nasal structures, etc.) predisposes to epistaxis, in all probability from the creation of a more or less complete vacuum behind the seat of obstruction. The writer has seen very alarming hemorrhage occur from such a condition, the blood flowing into the stomach and air-passages and leading to the suspicion of pulmonary disease. This is probably also the explanation of many cases of so-called "spontaneous" epistaxis. The same is true in regard to obstruction in the retro-nasal space, closure of the posterior nares plus the consequent congestion of the nasal passages, and the altered relations in regard to atmospheric pressure, conditioning the predisposition to hemorrhage. The writer has observed, on microscopic examination, quite extensive hemorrhages into the cavernous tissue in long-standing cases of hypertrophic catarrh. The extravasation may take place into the meshes of the cavernous body, or between it and the mucous membrane. When the bleeding has been slight, the only macroscopical evidence of its existence will consist in minute extravasations (ecchymoses) or capillary apoplexies. Usually the nasal mucous membrane is intensely hyperæmic. The hemorrhage generally arises from solution of continuity of the membrane at isolated spots or over circumscribed areas, rarely, if ever, from the whole surface of the nasal fossa.

It is a matter of common experience that various forms of excoriated mouth depend upon a disordered stomach, the so-called herpetic diathesis, etc., but occasional cases occur which cannot be referred to these conditions, whose dependence on hypertrophic nasal catarrh must be inferred from their disappearance with the cure of the nasal affection. Several factors are probably concerned in their production, among which mouth-breathing plays an important part, and, possibly, the disordered condition of the stomach occasioned by the nasal discharge.

Considerable interest attaches to the question of unilateral obstruction, as, for example, in many cases of deflection of the nasal septum. Not only is one nostril obstructed, the other remaining normal and becoming the vicarious channel through which respiration is accomplished, but the anatomical relations of both are changed. In the one case narrowing of the nasal passage results; in the other, abnormal dilatation of its cavity. The evils to which the former gives rise may be referred to two classes of effect, —viz., to the results of pressure and to those of mechanical obstruction. The former lead to atrophy, dislocation, or ulceration of the opposing structures. It is easy to conceive how this may happen, and that this is precisely what occurs is readily demonstrable by dissection.

The symptoms of advanced nasal obstruction have been well described by Meyer and others. The pallid countenance assumes a dull, stupid expression, and the cheeks become flabby from elongation of the naso-labial sulci. The mouth is kept open, the lower jaw depressed; the gums are fissured and cracked, and saliva dribbles from the mouth. This often leads the parents of the child to connect the stupid countenance and deafness with imbecility. Some writers call attention to the unusual prominence of the front teeth as a symptom of obstruction in the naso-pharynx, and Michel observed in a girl, aged eight years, the inner canthi of the eye so depressed that her countenance wore a strange, Chinese appearance. Deafness and tinnitus are nearly always present. Neuralgia is common. Taste is impaired. The nasal discharge is profuse, excoriating the nostrils, filling the pharynx, preventing sleep, and provoking suffocating attacks. These symptoms, together with constant snuffling, are well marked among children, and react most powerfully upon the general health. Several of my patients complained of a heavy dragging sensation in the back of the nose, which they compared to the presence of two heavy weights hanging into the throat. (The condition found was bilateral turbinated hypertrophy.) Later in life the nostrils became abnormally narrow, from arrested development or collapse of the alæ nasi. The speech becomes nasal, the tone of the voice dull and "dead" (Meyer). The tone is furthermore weakened and rendered indistinct by the interference with the motility of the soft palate from the presence of tumors and hypertrophies of the turbinated bodies. Obstruction in the nasal fossæ (deflected septum, polypi, etc.) prevents the free passage of the voice and diminishes correspondingly the force of the tone.

Differential Diagnosis.—Pronounced nasal obstruction in young children is generally post-nasal,—that is to say, in a large majority of cases the obstructive lesion will be found in the retro-nasal space. In a child suffering from impeded nasal respiration or symptoms of an ordinary non-suppurative otitis media, or both, if the forceps be introduced, without preliminary inspection, into the nasal pharynx, a mass of adenoid growth will generally be found in its grasp upon withdrawal.

Care should be taken not to confound true obstruction with the false obstruction so often caused by accumulated and inspissated secretion, crusts, foreign bodies, etc. The nostrils should be thoroughly cleansed, and, if necessary, the turbinated bodies contracted by means of cocaine, before inspection of the parts is undertaken. More or less permanent obstruction of the nasal fossæ may be conditioned by swelling of the turbinated tissue, brought about either as the result of collateral engorgement from obstruction in neighboring parts, as, for example, in the case of post-nasal growths, or as a reflex phenomenon dependent upon reflected irritation from a distant or an adjacent organ. This latter condition is especially true of the ear, the teeth, and the gastro-intestinal and genito-urinary tracts. In obstruction of reflex origin one or both nostrils may be affected.

One of the chief difficulties in the differential diagnosis of nasal obstruction is the separation of the hypertrophic conditions of the turbinated bodies from various outgrowths, etc., of the intra-nasal structures, and to this particular attention should be given.

The hypertrophied nasal membrane may be mistaken for a polypus, or, if situated on the septum, may be confounded with a deflection or outgrowth of that structure. The ordinary gelatinous polypus is much paler in color than the hypertrophied membrane, and presents usually a peculiarly brilliant reflection of light, which often leads to its detection in the upper and deeper portions of the nostril when no distinct growth is visible or suspected. Occasionally a somewhat similar reflection is observed in the hypertrophied, or even normal, membrane of the deeper portions of the illuminated structures, and may be mistaken for a growth. The red color of the hypertrophied membrane is usually uniformly distributed, while in the gelatinous polypus the enlarged vessels are thrown out in bold relief against a dull white or pale pinkish background. The vessels of the polypus, moreover, are seen to run from above downward, while on the turbinated bodies the direction is more commonly horizontal. The common mucous polypus is distinctly pedunculated, and its motility may readily be determined by the probe or nasal sound; or, if firmly impacted between the walls of the nostril, its pedunculated character may be brought into view and its motility demonstrated by the artificial contraction of the turbinated tissues by means of cocaine. The application of this drug to the membrane will cause mere puffiness of that structure to disappear, while it only markedly affects the color of a polypus. By closing the mouth and forcibly expelling the air through the nose, dislodgement and forward displacement of a distinctly pedunculated polypus will occur, while an hypertrophied membrane remains unmoved.

The gelatinous polypus is intensely hygroscopic, while the same amount of moisture will not necessarily affect the nasal membrane. Pressure on a polypus with the probe communicates to the hand the sensation of a soft, fleshy body, and when some force is used it bleeds readily, and also imparts a sort of crackling sensation to the finger of the operator. The hypertrophied membrane, on the other hand, though often yielding and resilient, has a hard bony foundation, which is readily detected upon pressure. Nasal polypus—and this is especially true of the fibrous or fibro-cellular variety—is often, though by no means always, confined to one nostril; while in the hypertrophic stage of catarrh both cavities are, to different degrees it may be, usually obstructed.

In hypertrophy the difficulty in respiration is principally in inspiration, while in polypus expiration is the act most commonly interfered with, the growth often acting as a ball-valve. A large polypus, or numerous small polypi, usually produce complete obstruction to both inspiration and expiration; in hypertrophy the air-way is rarely completely occluded. Unless the hypertrophy is very great, the voice is less distinctly nasal than when the nasal fossa is filled by a polypus. Polypi generally develop or spring from some portion of the middle meatus, while the most common seat of hypertrophy is the lower turbinated bone, and principally its posterior portion. Hemorrhage from one nostril was formerly considered diagnostic of polypi; but in our present knowledge of nasal hemorrhage in its relation to intra-nasal inflammation this test can no longer be relied upon.

In the posterior rhinoscopic image a mucous polypus can generally be distinguished not only by its glistening appearance, but also by its position, filling or obstructing the dark spaces of the meatuses described above.

The confusion of a hard, sessile, fibrous polypus situated in the deeper portions of the nostril with an hypertrophied condition of the turbinated bodies is sometimes a difficult matter to avoid; and this becomes more easily intelligible when we consider the anatomical mode of origin of many of these growths. The differential diagnosis between a posterior hypertrophy and a fibrous polypus, either originating in the nasal cavity or springing from the posterior ends of the turbinated bones and septum, is occasionally perplexing, and some confusion has arisen in the separation of the two conditions. Indeed, my examinations show that not a small proportion of the so-called fibrous polypi of this locality are nothing more nor less than enormously hypertrophied and pendulous turbinated bodies. It should also be remembered that now and then true fibromata with sessile bases on the anterior naso-pharyngeal wall may, instead of going downward, send one or more prolongations into the nasal fossæ and thus further complicate the diagnosis. A mistake may, however, be prevented by recalling the diagnostic appearances of the hypertrophied bodies posteriorly, and by attention to the fact that a fibrous polypus presents either a smooth or a distinctly lobulated appearance. Surgeons of wide experience in these matters will doubtless recall cases in which the fibrous polypus, originating by one or more pedicles in the nasal and accessory cavities, has sought the direction of least resistance, and presented at the posterior nares as a hard, immovable mass easily mistaken for an hypertrophied turbinated body. When we consider the mode of origin and growth of nasal polypi, and the manner in which they often fill the meatuses and destroy the normal anatomical relations of the parts, it is not surprising that, in the limited rhinoscopic picture, mistakes of this kind now and then arise.

On either side of the septum posteriorly are found, in the normal condition, two bulging hemispherical bodies, which are composed of erectile or contractile tissue, and care should be taken not to confound them with either polypi or true hypertrophy. In this climate it is rare to find the posterior nares in a condition which altogether meets the requirements of absolute anatomical and physiological perfection. The posterior ends of the inferior turbinated bodies are very frequently of an unnatural color and uneven shape, and this apart from any inconvenience to the individual. The novice, therefore, should hesitate before operating on such cases simply because the posterior nares do not present the appearances seen in diagrams and anatomical plates.

Cystic tumors of the posterior nares are exceedingly rare, but it would be well to bear their possible existence in mind in a doubtful case. Exostoses and hypertrophic enlargement of the turbinated bones themselves, and various malformations of the bony framework of the nasal fossæ, either congenital or acquired,—as, for example, in the altered anatomical relations of the nasal chamber left after necrosis and the expulsion of diseased bone,—so alter the normal appearance and position of the parts that it is well to call the attention of the beginner to their possible presence.

To the careful observer the confusion of hypertrophic inflammation of the septum with deflection or enchondrosis of the area ought not to happen; but, at the same time, it should be remembered that distinguished surgeons have made this mistake, and subjected patients to needless pain by bungling attempts at removal.

Hæmatomata and abscesses of the septum are sometimes met with, but are readily recognized.

Prognosis.—The prognosis will depend, of course, upon the cause and the facility of its removal, and upon the amount of structural injury already done to the nasal tissues and to those organs directly or indirectly affected by the destruction. In general, it may be written down as good. The removal of a nasal obstructive lesion cannot fail to relieve, even if it fail to cure; and it is in this field that some of the most brilliant triumphs of special surgery have been achieved.

REFLEX COUGH.

By ALEXANDER W. MacCOY, M.D.

Cough is a modified expiration. It is one of the most common and striking symptoms of many affections. In some instances cough is a very grave symptom; in others, it is one of the most harassing and difficult to ameliorate. On the other hand, a cough, as a symptom, may be out of all proportion to the gravity of the disease, the pathological basis of it being of the most trifling nature. Taken as a symptom of disease, it is treated as such to a greater extent than any other symptom except that of pain. The appreciation of cough as a symptom is greatly increased in value by certain characteristics, certain sorts of cough being pathognomonic of certain diseases. Often it is only one of many expressions of morbid states, without individuality and of no peculiar significance.

The varieties of cough are usually expressed by the terms hemming, hacking, hoarse, metallic, stridulous, aphonic, etc.; the cough may be short, sharp, paroxysmal or suffocative. The irritation may be expressed by a mild hemming or by a severe and grave suffocative attack. It may be dry or loose, with all the varying modifications between these extremes.

The cough-centre in the brain is said by Kohts to lie "on each side of the raphé in the neighborhood of the ala cinerea." Coughing is produced by stimulation of the sensory fibres of the vagus distributed to the mucous membrane of the larynx, trachea, and bronchi.[1] These cough-areas are such by reason of a freer distribution of the sensory fibres of the vagus to these particular locations. It will be evident that stimulation of fibres of the vagus in any part of its distribution may give rise to cough. This possibility enables us to explain the rationale of cough which is produced outside of the respiratory areas above mentioned, and to which we give the name *reflex* cough. It is difficult always to explain the rationale of reflex cough through the medium of the vagus, because of the complex character of the nerve. If we for convenience—arbitrary though it may appear—designate all cough-production *outside* of these cough-areas in the respiratory tract as reflex cough, we can elucidate the subject more easily. The act of coughing is always reflex, but the production of cough *outside* of what we may be allowed to term normal cough-areas must be considered as more correctly reflex. This brings us to our subject-matter,—the regions near to or remote from the areas in the larynx, trachea, and bronchi where cough may be produced.

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[1] Stoerck, of Vienna, does not consider that irritation of the smaller bronchi or accumulation of mucus in them is causative of cough, but believes that the cough-areas or "cough-spots" are situated higher up in the respiratory tract,—viz., in the inter-arytenoid fold, the posterior wall of the larynx and trachea, the under surface of the vocal bands, and the bifurcation of the trachea. These observations of Stoerck have been confirmed by Lennox Browne, of London, and others.

The region first in importance and where reflex cough arises most frequently is undoubtedly in the nasal passages. Nasal cough is a clinical entity; yet not long since such a possibility of production would have been considered a fantasy. Of late years the nasal reflexes have been exhaustively studied by investigators in rhinology, and the wonderful revelations incident to these investigations have enabled us to appreciate the far-reaching and complex character of the influences emanating from this sensitive region. The portion of the nasal chambers which we designate as the "respiratory tract," in contradistinction to the upper region to which the term "olfactory tract" is given, is that in which the reflex acts of coughing arise. The most sensitive parts of this respiratory tract are found where erectile tissue is most abundant, and particularly over the posterior portions of the lower turbinated bodies and septum. Other portions of the nasal chambers may give rise to cough under stimulation, but much less frequently than the posterior portions. This respiratory tract is where the catarrhal affections are chiefly found, and, owing to the unique vascular and free nervous supply and the large distribution of sympathetic fibres, it produces many clinical features peculiar and interesting.

By far the most common pathological state in which cough is produced is that of catarrhal inflammation, in the form either of acute coryza or of chronic hypertrophic rhinitis. Under such circumstances we have all the conditions active for the reflex manifestations. With the varieties of reflected irritation we have nothing to do excepting that of cough,—nasal cough. In the inflammatory conditions the sensory disturbances are readily induced, and cough excited either from hyperæmia, hypertrophy, or vasomotor disturbances,—from irritants without, or from internal excitants such as secretion or contact of swollen tissue, etc. It is a well-known clinical fact that a small pledget of cotton or a delicate probe introduced into the nasal chambers, in contact with certain areas and in certain subjects, will cause a reflex act expressed by a .cough. This cough can be kept up an indefinite period if the stimulation be continued. In many cases stimulation is expressed by the act of sneezing; this is generally produced in areas outside of the sensitive spots situated over the turbinated structure. The production of nasal cough is of so great interest and clinical value in affections of children that one ignoring it, or neglecting to appreciate its true position in the successful management of many affections of childhood, will often find his most vaunted remedies of no avail.

One of the most frequent and troublesome reflex coughs met with in children is the "night cough," a cough of nasal origin. Vogel speaks of it as "a periodic nocturnal cough." He believes it to be of nervous origin, but has failed to appreciate that it has a pathological basis in the nose and that it is of a reflex kind. Nocturnal cough in an infant or child, without pulmonary implication, occurring towards midnight, the child being in the recumbent position, is almost certain to depend upon a catarrhal inflammation seated in the nasal passages or naso-pharyngeal cavity. The manner of its excitation is as follows. The recumbent position is the most prominent factor in its production. After the child has been asleep for several hours, an accumulation of secretion in the nasal chambers takes place: turgescence of the posterior erectile tissue will be present. In the erect position this accumulation would be expelled from the nostrils anteriorly, or swallowed if it escaped posteriorly; but while lying down asleep, with functions in abeyance, it will naturally take the direction of gravity and lodge in the posterior nares upon the most sensitive areas, and, from contact alone or upon movement of the mucus, produce an irritation sufficient to cause a cough. This cough is short, dry, and irritative, most persistent and intolerable. If this secretion escapes into the naso-pharyngeal space and passes downward, it may produce another attack of cough. If the secretion gradually slides downward, it soon reaches the posterior portion of the larynx and lodges in the inter-arytenoid fold, where we have a true cough-area, and, under such circumstances, will produce an irritative cough which may continue for hours. When this mucus is expelled, the child falls asleep, and no further cough ensues until the following night, when there is a repetition of all the phenomena. So long as the coryza continues, the cough may be produced.

The rationale of the production of cough in this way can easily be verified by directing investigation to this region, and a cure can be speedily effected by addressing the therapeutic measures to the nasal chambers. In 1885, in the *Medical News*, I wrote a short paper upon the subject of "Night Cough in Children." Since that date I have seen many cases of cough produced in this way, and it has been my good fortune to have relieved many obstinate cases.

Several years ago the nasal symptoms shown in attacks of whooping-cough led me to suggest to Dr. J. M. Keating that there might be more nasal origin for such cases than was commonly supposed. During an epidemic of whooping-cough at the Philadelphia Hospital at that time, this suggestion was made use of, and the nasal passages of the children affected were treated with mild sedative sprays and antiseptics. The results were very satisfactory. Since that time the practice has been somewhat in vogue, and with fairly good results. The peculiar clinical fact that many paroxysms of whooping-cough end with a *sneeze* first drew my attention to the possible nasal origin of such paroxysms and caused me to make the suggestion.

Foreign bodies in the nasal passages may give rise to cough when great irritation is produced by their pressure (one-sided discharge, with occlusion, fetor, etc.). The form of atrophic nasal catarrh, or fetid catarrh, occasionally causes a nasal cough. An interesting case has been reported by Dr. White, of Richmond, Virginia. In such a case the stimulation is probably produced by the great accumulation of dried secretion acting as a foreign body. Relief is effected by getting rid of the pent-up and desiccated mucus by the use of alkaline washes combined with a good disinfectant.

Nasal polypi are not found in young children: in adults they occasionally give rise to cough. The different conditions of enchondroma and exostoses and nasal deflections are frequent in childhood, and may produce cough from pressure and by occlusion. In the naso-pharynx (which should be considered as a part of the nasal tract) we have in childhood several hypertrophic changes from which cough may be produced. A rather frequent condition found in children is hypertrophy of the pharyngeal tonsil, called the tonsil of Luschka, and also spoken of as adenoid tissue at the vault of the pharynx. This term, adenoid tissue, however, embraces more than the enlargement of the pharyngeal tonsil itself, including hypertrophy of *all* the lymphoid tissue situated at the vault of the pharynx. Hypertrophy of this structure, if considerable, not only leads to increased redness and secretion, but also causes obstruction to nasal breathing, and, if enlarged sufficiently to occlude the posterior spaces, mouth-breathing follows, with its train of evil consequences. From pressure, the hypertrophied tissue may give rise to congestion of the nasal passages and of the lower naso-pharynx and fauces, increasing the sensory excitability. A very slight irritant furnishes the necessary stimulation for the production of reflex disturbances, in some instances expressed by cough. To this naso-pharyngeal region we have certain fibres of the vagus distributed.

Follicular pharyngitis, acute and chronic, often seen in children, gives rise to cough. This is generally a disease secondary to a chronic nasal catarrh. These enlarged follicles are often not only painful but also very susceptible to irritation. Frequently the passage of air over these inflamed structures will produce a short, dry cough.

Hypertrophy of the tonsils, so common in childhood, with many other symptoms produces a cough which at times takes the form of suffocative attacks and is paroxysmal. An elongated and inflamed uvula sometimes causes cough in children by mechanical irritation of the base of the tongue, though this is not a frequent condition in early life. Enlarged lingual papillæ or lymphoid tissue situated at the base of the tongue, when present in children, occasions a most obstinate dry cough, when this hypertrophied tissue interferes with the play of the epiglottis and irritates its lower surface. This condition, too, is in my experience infrequent in children.

Ear-cough is not uncommonly present in certain conditions of the auditory meatus and membrana tympani. According to Dr. J. C. Blake, the ear-cough is produced in the following manner: "the irritation of the sensitive fibres of the auriculo-pneumogastricus, distributed in the meatus and to the membrana tympani, is reflected along the motor fibres of the superior laryngeal nerve, exciting in the larynx the act of coughing by causing contraction of the crico-thyroid muscle." This ear-cough can be produced by irritation set up in the auditory meatus by accumulation of wax, when the cerumen is unusually dry and loosely confined in the ear. Motion of the jaw will cause movement of this dried wax, and by titillation of these parts produce a short, dry cough. There are a number of cases in literature where the cough was persistent from dry wax in the ear, but was quickly relieved as soon as the wax was removed. An aural speculum placed in the ear *cold* will sometimes cause a cough. Foreign bodies in the ear are another cause of cough: some interesting cases are on record where the cough disappeared like magic upon removal of the foreign body. Necrosed bone at times acts as a foreign body, producing ear-cough. Sudden arrest of a chronic discharge from the middle ear has produced cough, which ceased when the flow of pus returned. This reflex phenomenon of ear-cough is of sufficient frequency in childhood to render it necessary that a careful inspection of the ear should be made in all obscure cases where the cause cannot be found in the respiratory organs.

Cough produced by irritation of the fibres of the vagus distributed to the alimentary canal has been called stomach-cough, and the term may be considered traditional. Stomach-cough probably exists, but we doubt if there is just ground for the loose way in which this term is used in explanation of many obscure cases of cough. It is probable that many of these cases have a real pathological seat higher up, either in the respiratory organs or in the auditory canals. Foreign bodies retained in the stomach or in other portions of the canal have been mentioned as causes of a cough which has been promptly relieved by the expulsion of the foreign substance. Undigested or indigestible articles of food remaining in the stomach have produced cough, and the reflex phenomenon disappeared only when the stomach had ejected its contents. There are fairly well authenticated cases where the expulsion of tapeworm, lumbricoids, and other parasites from the alimentary tract has caused the immediate arrest of a persistent and vexatious cough.

Umbilical protrusion has been reported as the exciting cause of violent cough in an infant four weeks old. Replacement and compression promptly relieved all the symptoms.

Basilar meningitis and abscess or tumor of the cerebellum have at times a short, dry cough as an accompanying symptom, and, when present, it should not be allowed to mislead one in diagnosis.

There are probably many other anomalous conditions which give rise to cough, and, when we consider the well-known susceptibility of childhood to impressions of every sort, it need not be thought remarkable that the causes leading to cough of a reflex nature should be so various and so complex. Due weight should be given to these anomalous features, and in making our observations we should be ready to believe that it is the unexpected which always happens.

EPISTAXIS.

By ETHELBERT CARROLL MORGAN, M.D.

Etymology.—From the Greek ἐπιστάζω, to "distil:" a "dripping."

Synonymes.—Nosebleed; Latin, Rhinorrhagia, Hæmorrhinia, Hæmorhinorrhagia, Choanorrhagia, Hæmorrhagia narium; French, Épistaxis, Saignement du nez; German, Nasenbluten; Italian, Epistassi.

Definition.—Bleeding originating in the nasal passages.

Explanation.—The application of the word epistaxis is limited, by the writer, to those hemorrhages originating in the nasal cavities proper. Hippocrates appears to have applied the name to bleeding of the cutaneous or mucous surfaces of the nose, whilst Vogel and Pinel used it to describe nasal hemorrhage due to any cause. The mere escape through the nose of blood flowing from the maxillary, sphenoidal, ethmoidal, or frontal sinuses, from the middle ear, from a cranial fracture, or indeed from the lungs or stomach, does not constitute a true epistaxis, but rather a hemorrhage from the regions mentioned, having its channel of exit through the nasal passages. Rhinology demands a definite and explicit nomenclature, whose ultimate purpose is the proper description and location of pathological phenomena, as well as the avoidance of indeterminate and perplexing terms. Whilst the writer much prefers the words rhinorrhagia and nosebleed to the term epistaxis, as expressive of the condition under consideration, custom renders retention of the word epistaxis advisable. The three terms, therefore, will be used synonymously in this article.

The escape of blood from the nostrils, therefore, may or may not constitute what in the writer's opinion is an epistaxis, conformably to the definition above given.

History.—The early medical writers fully describe nosebleed, and accord it an important part in connection with the progress of many morbid conditions. It was generally taught that nosebleed could, under varying circumstances, be the precursor of death or the harbinger of convalescence. We find both Hippocrates and Galen[1] estimating epistaxis as a valuable prognostic factor,—the latter rendering himself famous by gravely informing a patient that his sufferings would terminate in death by a critical hemorrhage from the right nostril.

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[1] Galen (Claudius), De Crisibus, lib. iii. cap. 3, 16mo, Lutetiæ [1528].

Galen knew that the application of a large cupping-glass to the hypochondria would arrest nasal hemorrhage. Verneuil[1] has recently (1887) described his experience in the treatment of certain forms of epistaxis by counter-irritation over the region of the liver, but, to his great surprise, finds that Galen anticipated him. The occasional and minor hemorrhages of childhood, which usually cease spontaneously and are of slight import, received attention at the hands of the Arabians, the Greeks, and the Romans, few conditions having been more fully appreciated or more accurately described.

Man alone among animals is subject to this form of hemorrhage. Veterinary surgeons, however, state that violent exercise and strain may cause nosebleed in the horse, and it is recorded that one great racer bled every time he ran.

Celsus fully descanted upon what is to-day denominated vicarious epistaxis, demonstrating that the so-called supplementary hemorrhages were properly recognized and appreciated in early medical history.

Fabricius of Hilden mentions the destruction of varicose veins and the reduction of cutaneous swellings as fruitful sources of epistaxis. Peyer maintained that the sudden stoppage of the bleeding in plethoric children often leads to fulness of the head, flushing of the face, and pains in the ears and forehead. Aretius was aware of the connection existing between epistaxis and the various fevers. Both Hoffmann[2] and Tulpius record instances where engorgement and cirrhosis of the liver were attended by epistaxis.

Morgagni[3] states that a very fatal epidemic, characterized by bleeding from the nose, is recorded to have occurred in Etruria and Romandiola in the year 1200. Gilchrist mentions epidemics of nosebleed, but it is evident that this accident was only symptomatic of some malignant fever which then prevailed.

Ancient writers also accorded to that frequent and striking symptom epistaxis the minutest clinical investigations, which resulted in their formulating a host of diagnostic and prognostic aphorisms, often as true as they are interesting.

There are really three periods in the history of epistaxis, the first extending up to the time of the introduction of Belloc's canula (1804?), the second thence until the employment of rhinoscopy (Czermak, 1858), and the third from that date to the present time. The two latter periods have produced much of value and interest concerning the pathology, diagnosis, and treatment of this condition, but all recent writers (including myself) have found the classic and exhaustive work of Cloquet,[1] published in 1821, *facile princeps* on all the subjects therein treated.

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[1] Verneuil, Traitement de certaines Épistaxis rebelles, Bull. Acad. de Méd., Paris, 1887, 2me Sér., xvii. 489–505. Also, Semaine Méd., Paris, 1887, vii. 168.

[2] Hoffmann (F.), Med. Rationalis System., Opera Omnia [etc.], Genevæ, 1740, 200.

[3] Morgagni, De Sedibus et Causis Morborum [etc.], Epist. 14, No. 25, Patav., 1765.

Etiology.—There are very few persons who have not at some period of life, especially during childhood, experienced nosebleed, and so frequent and insignificant is the flow during this period of life that it deserves but passing notice.

Hippocrates was satisfied to designate epistaxis by the unqualified term "hæmorrhagia," regarding it as by far the most frequent of hemorrhages as well as the most important of symptoms. It is generally, in childhood, primarily dependent upon active congestion, and may then be denominated nature's favorite mode of bloodletting. The great vascularity of the lining membrane of the nasal cavities and surfaces of the turbinated bodies favors the occurrence of nosebleed, rendering it the most easily produced as well as the most frequent of local hemorrhages.

The causes of nosebleed are extremely varied, and a correct etiological classification would simply include an enumeration of local and constitutional disturbances coincident with, or productive of, this symptom. Numerous etiological classifications have been proposed by writers upon epistaxis since the able work of the learned Hippolyte Cloquet, but no genuine advance has been made towards a simplification of the subject.

Cloquet[2] divided epistaxis into three classes: 1st, the traumatic; 2d, the plethoric (active); 3d, the adynamic (passive). Jaccoud divides it into (1) traumatic or ulcerative; (2) that resulting from disease of the blood-vessels; (3) mechanical, (*a*) active, (*b*) passive; (4) adynamic. Watson[3] classifies it into (1) traumatic or lesional, (2) plethoric or active, (3) adynamic or passive. Bouty[4] maintains that it is well-nigh impossible to classify and limit the causes producing nosebleed, for the reason that a majority of the cases appear to be due to individual peculiarities, each case demanding a special analysis.

Nosebleed occurs (1) from the so-called "hemorrhagic spots,"—the "predilection points" of Baumgarten; (2) as a symptom of various intranasal diseases; (3) as a symptom of general disease; (4) as a result of vaso-motor influence. Having a nasal hemorrhage to deal with, we first look for the usual local causes, such as traumatism, erosions, ulcerations, necroses, tumors, particularly vascular polypi, rhinoliths, or congestions of the nasal mucous membrane; but, failing to find these, we should examine the internal organs, and recognize existing or approaching eruptions and infectious diseases, dyscrasiæ, and diatheses, as well as vaso-motor disturbances and other nervous derangements.

Clinical observations of rhinorrhagias occurring during childhood emphasize the fact that in many instances the loss of blood occasions no appreciable injury, and may, in many cases, be salutary.

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[1] Cloquet (H.), Osphrésiologie, 8vo, Paris, 1821.

[2] Loc. cit.

[3] Watson (W. Spencer), Diseases of the Nose, 8vo, London, 1875.

[4] Bouty (J.), Contribution à l'Étude de l'Épistaxis, 4to, Montpellier, 1884, No. 13.

Rhinoscopic examination of the nares of these children, shortly after hæmostasis, fails to reveal any permanent or marked lesion, a simple dark line or point indicating the source of the previous hemorrhagic flow. Statistics derived from a large number of cases show that the bleeding, in at least seventy-five per cent. of these children, takes place from fixed points or areas within the nares, conveniently designated "hemorrhagic points," "points of predilection" (Baumgarten), or points of least resistance. In a healthy child, with normal nares, the temporary congestion of the cavernous sinuses leads to a break or fissure in their walls, and the consequent escape of blood. The local lesion and the constitutional disturbances resulting from this natural process of depletion are insignificant, and the phenomena may, in the writer's estimation, be considered as physiological or normal in the class of cases under consideration.

The condition observed in children who apply to the rhinologist for relief from physiological or normal rhinorrhagia is likewise noted when the hemorrhage is symptomatic of nasal or general disease,—that is, a predilection for certain points from which the bleeding occurs. The existence of intra-nasal inflammation, ulceration, etc., is a fruitful cause of nosebleed, especially in plethoric persons. Blowing or scratching the nose, vomiting, coughing, sneezing, and the inhalation of irritating powders, gases, etc., may result in engorgement of the nasal mucous membrane, capillary rupture, and epistaxis. The same may be said of blows on the nose or surrounding parts, surgical injury, fractures, contusions, cauterizations, or the careless introduction of the Eustachian catheter, Politzer bag, or nozzle of the spray-apparatus.

The nasal septum is frequently denuded of the thin and delicate mucous membrane with which it is invested, in children who pick their noses with their finger-nails: an obstinate erosion, giving rise to repeated attacks of nosebleed, may thus result.

Cases are reported in which the inhalation of certain odors produced nosebleed, the most remarkable being that of Jean de Querceto, secretary to Francis the First, who upon smelling an apple always had a hemorrhage from the nose. Certain powdered drugs, such as scammony, jalap, hellebore, and ipecacuanha, when inspired, have been known to cause nosebleed.

Frequently it is traceable to exposure to cold or immoderate heat, or to diminished atmospheric pressure, as is frequently observed in mountaineers and others during the ascent of high mountains.

Nosebleed also frequently results from the suppression of an exanthem or of the perspiration, and from irregularities in the menstrual or hemorrhoidal flow or other customary depletory hemorrhages.

It is well known that rhinorrhagia is habitually associated with various hepatic, renal, cardiac, splenic, and pulmonary affections; and in scurvy, purpura, erysipelas, diphtheria, malarial and malignant fevers, it is an ordinary accompaniment. In typhoid fever, in connection with other symptoms, it is considered pathognomonic, and it is a symptom of frequent occurrence in threatened uræmic attacks.

Age.—Rarely observed in the new-born or suckling, nosebleed becomes more common as the child advances towards puberty. In old age, senile atrophy of the pituitary membrane and impermeability of the vessels are said by Meyrignac[1] to explain the rarity of epistaxis. According to Baginsky,[2] eleven per cent. of school-children have epistaxis. Childhood predisposes to epistaxis, as do also a morbid condition of the capillary vessels and previous attacks of bleeding.

Bartholin mentions the aversion of certain children in a family to butter and cheese. When coaxed and bribed to eat of these articles, they were invariably seized with vomiting and nosebleed.

Sex.—Nosebleed is observed more frequently in boys than in girls. Towards the age of puberty it may be vicarious in the latter. Menstruation, and a life of less exposure, sufficiently explain the comparative infrequency of epistaxis in females.

Sir Thomas Watson says, "The readiness with which the mucous lining of the nasal passages pours forth blood is familiar to the experience of every school-boy, who often wipes a bloody nose."

Side Affected.—There is little to be said on this subject, further than that the hemorrhage would seem to occur as frequently from one side as from the other,—at least, in simple, uncomplicated cases.

Epistaxis during the course of hepatic disease was said by Galen[3] to take place from the right nostril, whilst in splenic and cardiac disease the blood invariably flowed from the left. When, however, this order of things was reversed and the bleeding occurred from the left nostril in hepatic or from the right in splenic disease, it was regarded as a bad omen. It is superfluous to add that these hypotheses are in no way verified by clinical observation.

It is not always possible to discover from which side the hemorrhage proceeds, for when it is very copious the blood may flow from the unaffected side as well as from the other, and may even flow from the throat.

Predisposition, Idiosyncrasy.—There is a marked and constant tendency to nosebleed in some individuals upon the slightest provocation, while others seem to enjoy absolute immunity. Whilst in children rhinorrhagia is usually primary and traceable to active congestion, in old age it is symptomatic; it is then venous, and is the result of passive congestion.

Those rare cases traceable to the odors of fruits, flowers, and food constitute individual peculiarities.

Heredity.—A tendency to epistaxis is occasionally hereditary. Families the subject of a hemorrhagic diathesis may likewise inherit a special tendency to nosebleed. Babington recorded a remarkable instance in which six females, like their mother, suffered from frequent nosebleed. The heredity in Babington's citation included three generations.

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[1] Meyrignac (C.), De l'Épistaxis considérée comme Hémorrhagie sénile. Thèse, 4to, Bordeaux, 1887, No. 48.

[2] Baginsky, Gerhardt's Handb. d. Kinderkrankheiten, Tübingen, 1887, vol. i.

[3] Loc. cit.

Violent Exercise.—Running, jumping, bodily fatigue, and strain from lifting are common causes. Alibert knew a very young lady who had nosebleed whenever she danced. Coitus has been known to cause it in persons of a sanguine temperament. A sedentary life, high feeding, and continence are said to favor its occurrence.

Mental Emotions.—Anger, fright, grief, and joy, by determining cerebral plethora may cause epistaxis. Undue mental application in the young and frail, as well as in the robust, and the reflex irritation resulting from the presence of intestinal worms, are also recognized as occasional causes.

Diagnosis.—The diagnosis of a discharge of blood through the nasal passages is easy, and it is only in those rare cases in which the hemorrhage is to be differentiated from that originating in the accessory sinuses that any doubt can arise. The recognition of bleeding from the sinuses, which in adults is a matter of the greatest difficulty, is in children practically impossible.

Whenever practicable, a careful anterior and posterior rhinoscopic examination, which is by no means easy in all children, should be made, and the bleeding point or points located.

This accomplished, the indications for therapeutic action become apparent, and their execution is more likely to be successful.

The nasal speculum of Duplay, or, in very young children, the oval otoscope of Gruber, will permit the proper illumination and examination of the anterior nares. The bloody coagula obstructing the view should be removed by injections or inspiratory efforts, after which the superior meatus should be mopped with absorbent cotton: failing to find the seat of hemorrhage, the lower meatuses and turbinated tissues should be cleansed in the order and manner mentioned.

This examination will demonstrate whether an erosion, epithelial desquamation, ulcer, varicose dilatation, angioma, or polypus is the source or cause of the bleeding. Kiesselbach noted the above causes in thirty-three out of his thirty-eight cases of rhinorrhagia.

Epistaxis in Diphtheria.—Hemorrhage is a frequent grave and fatal complication in the nasal variety of this disease, Parker Smith having lost twelve out of thirty children whose nostrils, by reason of tender age, it was impossible to tampon. The bleeding may take place at the very onset of the disease or occur at a later period, and is, according to Trousseau,[1] a most important symptom, and the forerunner of the development, upon the pituitary mucosa, of the characteristic false membrane. Nosebleed has long been regarded as a phenomenon of great gravity. De Heredia, one of the authors who described the great epidemics of malignant angina which prevailed in Spain at the beginning of the seventeenth century, says, "Malignam significationem praebet sanguis stillans e naribus. . . . Periculosissimus censetur sanguinis fluxus ex naribus aut ore." These hemorrhages may occur repeatedly during the day, either as a persistent oozing or as a copious and exhausting flow.

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[1] Trousseau, Clinique Méd., Paris, 1868, i. 389.

Statistics show a fatality of seventy per cent. in epistaxis of the initial period, and eighty per cent. in that of a late period, in diphtheria.

Epistaxis in Pertussis.—Epistaxis due to convulsive cough is a common concomitant of whooping-cough, and, if profuse or oft-repeated, contributes in a degree to the asthenia attending this disease. The bleeding may occur at each coughing-spell, or indeed during the intervals, which is a more serious complication. The blood may flow backward through the post-nasal orifices into the throat, be swallowed, and discharged per rectum in the form of dark masses, or it may be vomited during a paroxysm of cough. The latter occurrence has caused grave anxiety in the parents, by creating the suspicion that the blood was coughed up from the lungs or the stomach as the result of hemorrhage of those organs.

The congestion of the blood-vessels of the head resulting from the violent paroxysms of cough may also determine a rupture of the vessels of the conjunctiva in addition to the rhinorrhagia.

Epistaxis in Respiratory Diseases.—Nosebleed occasionally occurs in the course of pleuritis, empyema, pneumonia, emphysema, or fibroid induration of the lung. Interference with the pulmonary circulation may result in an epistaxis. In nasal hemorrhage occurring in pneumonia the blood may be pale or serous, the flow intermittent, and the pulse and respiration greatly accelerated.

Epistaxis in Hepatic Disease.—Clinical observation and the results of treatment prove that both acute and chronic disorders of the liver are important and frequent causes of obstinate epistaxis. Impeded hepatic circulation and alteration of the blood, together with increased vascular tension, may lead to the rupture of those intra-nasal capillaries weakened by local disease.

Colin[1] regarded secondary cardiac disease as responsible for the nosebleed accompanying hepatic affections. Harkin[2] maintains that an anæmia inducing a hemorrhagic diathesis results from changes produced in the blood by hepatic disease.

Epistaxis in Splenic Disease.—In nearly all affections of the spleen nasal hemorrhage is a common symptom,—so common that the ancients were wont, as are many of the laity to-day, to regard a repeated bleeding from the left nostril as pathognomonic of splenic disease. The causative relation existing between epistaxis and disease of the spleen, whilst usually marked, may in rare instances be a matter of doubt.

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[1] Colin (d'Alfort), Relations entre les Affections du Foie et les Épistaxis, Bull. Acad. de Méd., Paris, 1887, 2me Sér., xvii. 502.

[2] Harkin (A.), On the Nature and Treatment of Epistaxis, Med. Press and Circular, London, 1887, xliv. 215.

Hyperæmia, simple hypertrophy, and lardaceous degeneration of the spleen give rise to leukæmia and thus occasion epistaxis. Moreover, diseases of the spleen are habitually associated with exhausting maladies which show a tendency to epistaxis.

Epistaxis in Rheumatism.—As the result of arthritic and gouty affections epistaxis is by no means unknown, even among children. Those having a deposit, gravelly in character, in their urine, resembling that of gout and rheumatoid pains, may, as they advance in age, show a marked disposition to occasional epistaxis, which does not appear to produce asthenia.

The nosebleed of rheumatism, which occurs usually during the second stage of the disease, between the tenth and the fifteenth day, is slight, and may coincide with a marked amelioration of the general symptoms. From the frequency with which an epistaxis shortens the disease or diminishes the sufferings of the patient, it is esteemed a welcome phenomenon in rheumatism.

Epistaxis, in rare instances, has been known to occur as a critical accident, the result of the adynamic state of the patient, or as a precursor of serious cerebral complications.

Epistaxis in Tuberculosis.—Bouffé[1] thus defines this variety of nosebleed: "All hemorrhages appearing without known cause, most often spontaneously, and recurring frequently during an indefinite period, flowing drop by drop, ceasing spontaneously, and rarely requiring tamponing." He claims to have observed rhinorrhagia in seventy-five per cent. of his observations upon tuberculous patients, as a premonitory, concomitant, or terminal phenomenon.

The characteristics of premonitory epistaxis are its frequency, duration, quantity, and time of occurrence. These hemorrhages are often repeated during months and years; they may last five to ten minutes, flow drop by drop, and increase after the seventh year of life. In the female, the establishment of menstruation marks their starting-point.

Rhinorrhagia may occur as a concomitant of the thoracic lesions of tuberculosis, and may coincide or alternate with the hæmoptysis so common in that disease. Terminal epistaxis in the tuberculous has been variously attributed to pathological changes in the kidneys, to alterations of the blood, and to lesions of the pituitary membrane. That a well-defined tendency to ulceration of the air-passages exists in tuberculous subjects is certain, and the pituitary membrane appears to be especially susceptible to it.

Epistaxis in Eruptive Fevers.—Rhinorrhagia is often observed in measles as an initial symptom, and less frequently in scarlatina and variola, although in the latter disease it is justly regarded, particularly when it occurs conjointly with hemorrhages from other mucous membranes, as a symptom of great gravity. These hemorrhages are insignificant in measles, and gain prognostic import only when the child is profoundly anæmic or is covered with petechiæ, as in the hemorrhagic type of rubeola. Rhinorrhagia, as has already been said, may be symptomatic of suppressed exanthemata.

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[1] Bouffé (M. F.), Recherches sur l Épistaxis chez les Tuberculeux, 4to, Paris, 1877, No. 383.

In facial erysipelas the occurrence of rhinorrhagia usually indicates an extension of the disease to the nasal passages.

Epistaxis in Typhoid Fever.—Hippocrates, Galen, Sydenham, Willis, Hoffmann, Vogel, Pinel, Gilchrist, Van Swieten, and numerous other writers mention rhinorrhagia as occurring in grave fevers having all the symptoms of the typhoid of our day. The frequency and the character of the bleeding in enteric fever are greatly modified by the occurrence of that disease in epidemics, and by climate, temperament, and age. Rilliet and Barthez[1] noticed epistaxis in one out of five of their typhoid cases, and usually about the sixth day. Griesinger considers that epistaxis is most frequent and more abundant in the younger subjects of this fever. It has always been regarded as a diagnostic sign in typhoid fever.

Periodical Epistaxis.—Nosebleed sometimes assumes a periodical or intermittent character: in such cases it is generally associated with a well-defined malarial condition. There are, however, certain others in which the hemorrhage becomes, so to speak, habitual, and this without the coincidence of impaludism.

Vicarious Epistaxis.—Menstruation may be partially or entirely supplanted by a hemorrhage from the nasal mucous membrane, constituting what is generally denominated a vicarious epistaxis. The possibility of such bleeding is universally admitted by the profession, examples being commonly observed by the gynæcologist and the laryngologist. Vascular tension is greatly increased during and prior to each menstrual period, which may result in irruption of blood from some organ remote from the uterus; the nasal passage may in this manner be the seat of capillary rupture and consequent hemorrhage. It would appear that, in addition to amenorrhœa, there must be a simultaneous disturbance of the vaso-motor nerves or vessels in the particular organ from which the blood escapes, to produce vicarious hemorrhage.

Epistaxis in Masturbation.—The intimate relation or sympathy existing between the nose and the sexual organs was described in ancient medicine, and serves with the observations of to-day to explain a variety of rhinorrhagia hitherto ignored or overlooked. That rhinorrhagia is most frequent about the age of puberty and in children given to self-abuse seems well established. The sexual disturbance peculiar to this period in some children is followed by onanism, which in turn causes irritation of the genitalia, reflex turgescence of the cavernous tissues of the turbinated bodies, and possibly a consequent epistaxis. It is known that occasionally a slight stimulation of the nasal mucous membrane, of the skin or the eye, and even mental emotion, may, by reflex action, produce engorgement of the cavernous tissues. If the blood-pressure accompanying these engorgements exceeds the strength of the distended capillaries, their rupture, with that of their epithelial covering, results in epistaxis.

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[1] Barthez and Rilliet, Maladies des Enfants, vols. i. and ii., 8vo, Paris, 1861.

The coexistence of onanism and epistaxis has been described by Germain Sée and Rendu,[1] but to Joel we are indebted for the most exhaustive and conclusive article upon the subject.

Epistaxis following Surgical Procedures.—The operations which the rhinologist is called upon to perform in the nasal fossæ of children, such as the extirpation of benign growths, the removal of exostoses, the correction of deformities due to deflected nasal septa, etc., are generally unattended by serious hemorrhage. The employment of hæmostatics upon slight pretext after intra-nasal operations may delay the reparative processes; and the same may be said of tamponing. Although cocaine hydrochlorate is conceded to be of inestimable value in nasal surgery by greatly facilitating all operative measures, it is the firm belief of the writer that its use occasionally invites a secondary hemorrhage which is difficult to control.

Prognosis.—The gravity of a prognosis in epistaxis depends wholly upon the particular condition of which it is the symptom or complication, and all therapeutic intervention should be governed by the etiological factors of the case. The age and strength of the child, and the amount, frequency, and cause of the bleeding, should be considered in reaching a prognosis.

Rochoux says, "The numberless and varied causes to which epistaxis is attributable render this accident always identical in its apparent phenomena,—an affection quite different in its essential nature, and one demanding a varied treatment."

The prognosis in traumatic epistaxis is generally favorable, as the hemorrhage is slight and ceases spontaneously. Barthez, Rilliet, and Valleix, who have examined a great number of recorded cases of epistaxis in children, have failed to find a single one of primary epistaxis that has proved fatal.

The occasional and moderate discharge of blood from the nostrils, in health or in certain acute and chronic diseases, may be salutary: instances are recorded where meddlesome interference has resulted in serious consequences to patients (cases of Van Swieten, Hoffmann,[2] Portal, J. Frank,[3] Cazalis, and Watson). So firmly did the older writers believe in the beneficial results of natural depletion from the nasal membrane, in renal, cerebral, and hepatic disease and in certain eruptive fevers, that even the enlightened Hoffmann recommended and practised the production of artificial epistaxis.

Whilst in elderly persons epistaxis may indicate a tendency to apoplexy, obstruction of the cardiac or pulmonary circulation, or organic disease of the liver or numerous pathological disturbances of the internal organs, it is in children almost universally associated with a sanguine temperament, and is, therefore, of slight importance, calling for no interference. In plethoric subjects epistaxis may constitute a salutary form of local depletion, contributing to the relief of cerebral congestion, but if permitted to recur frequently and copiously the child may become weakened and anæmic. Cases of exhausting and fatal epistaxis do occasionally occur, either from neglect, or from persistency of the hemorrhage: so that the possibility of such an accident should be borne in mind. Rilliet[1] saw a fatal case (secondary) of epistaxis in a child of four years. S. D. Gross has seen five fatal cases of all ages, due to imperfect tamponing or to delay in operating until the patient was exhausted by bleeding. A fatal case of epistaxis is reported by Craft.[2] A girl who had never menstruated, but in whom the menstrual molimina were accompanied by copious nasal hemorrhage at intervals of six weeks, finally died from the resulting exhaustion.

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[1] Rendu, De l'Épistaxis chez les Enfants, Semaine Méd., Paris, 1884, Nos. 24–26.

[2] Hoffmann (F.), Med. Rationalis System., Opera Omnia [etc.], Genevæ, 1740, 200.

[3] Frank (J. P.), De Curandis Hominum Morbis, Mannheimii, 1807, lib. v. pars 2, 124.

As the rhinorrhagic child advances in age, a hemorrhoidal flow, hæmoptysis, or cerebral or other troubles, may replace nasal hemorrhage.

Sir Thomas Watson tersely presents the prognosis in epistaxis when he says, "Sometimes it is a remedy, sometimes a warning, sometimes really in itself a disease."

Pathological Anatomy.—The nasal mucous membrane is intensely congested, swollen, and of a bright red or purple hue, and may be studded here and there with ecchymotic areas. The extravasation of blood into the submucous and spongy tissues, and their consequent distention, result in partial or complete occlusion of one or both nostrils, which is relieved by the subsequent hemorrhage. Usually one or more ruptured vessels are visible, constituting the bleeding points, which a rhinoscopic examination often reveals during life. These bleeding points were known to Valsalva and Frank, and have been more fully described by Michel,[3] Little,[4] Lefferts,[5] and Baumgarten;[6] the latter furnishes some valuable data as to their most frequent position. They are limited to the lower or respiratory area of the nasal passages, and almost universally to the anterior portion of the septum. Among one hundred and thirty-six recorded cases of nosebleed in which the bleeding point was visible, one hundred and twenty-six were from the septum, eight from the inferior turbinated bone, and two from the floor of the nose.

An examination of the delicate pituitary membrane in childhood in a state of health shows it to be formed of a weak and very lax stroma, over which is a frail and delicate cylindrical ciliated epithelium, which affords feeble support to the walls of the innumerable vessels everywhere present.

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[1] Loc. cit.

[2] Craft (M. S.), Fatal Epistaxis, Trans. Miss. Med. Assoc., 1881, xiv. 104.

[3] Michel (K.), Die Krankheiten der Nasenhöhle, 8vo, Berlin, 1876.

[4] Little (James L.), A Hitherto Undescribed Lesion as a Cause of Epistaxis, with Four Cases, Hospital Gazette, New York, 1879, vi. 5.

[5] Lefferts (George M.), A Practical Point concerning Epistaxis, Medical News, Philadelphia, 1882, xl. 100.

[6] Baumgarten (E.), Die Epistaxis, Wien, 1886, p. 47.

The blood-supply of the inferior and olfactory regions is derived from the internal maxillary artery, and includes the spheno-palatine, branches of the infra-orbital, superior alveolar, palatine, and pterygo-palatine, as well as some branches of the supraorbital and some ethmoidal veins which are distributed mainly over the olfactive area. The artery of the dorsum and those of the alæ of the nose, the latter of which freely anastomose with the artery of the septum, complete the list of vessels from which the bountiful blood-supply of the nasal passages is derived. The intercommunication of the veins of the nose and the sinuses of the dura mater is well known.

Symptoms.—The onset or manner of appearance of epistaxis is extremely varied. There are often well-defined prodromes, but occasionally all premonition is wanting, the hemorrhage occurring suddenly. The premonitory symptoms, "molimina hæmorrhina," are frontal headache or pressure, flushing and congestion of the cheeks and face, itching of the nose, giddiness, vertigo, tinnitus aurium, and burning and hyperæmia of the ocular and palpebral conjunctiva, to which may be added pain in the temples and pulsations of the temporal or carotid arteries. The bleeding is, however, in the majority of children simply preceded by a sensation of dizziness, pressure in the head, or dryness, heat, tickling, and obstruction of the nostrils, and it not infrequently begins while the child is at play. To these symptoms may be added a desire to pick or blow the nose, the staining of the handkerchief in the latter act being often the first indication of the hemorrhage.

The blood may flow from one or both nostrils: from one when the bleeding point is in that naris, the hemorrhage moderate, and there is no perforation of the septum; from both nostrils when a copious bleeding from any source, filling the naso-pharynx, enters both posterior nasal openings, escaping anteriorly. Epistaxis, in children having a unilateral anterior nasal stenosis, takes place from the unobstructed nostril or naso-pharyngeal opening. Occasionally it is found that the blood has forced its way backward around the septum and escapes from the opposite nostril; and this may occur even in nares of normal dimensions.

In rare cases a considerable amount of blood may be discharged posteriorly through the naso-pharyngeal opening and swallowed. In very young children this hemorrhage may result in exhaustion before the discovery is made.

Duration.—Bleeding of the nasal mucous membrane generally ceases in from ten to twenty minutes, but in exceptional cases is protracted for hours or even for days.

Quantity.—The blood usually flows in drops from one nostril, "stillicidium sanguinis," or in a continuous stream, "rhinorrhagia." It should be borne in mind that there is frequently an exaggeration, as regards the amount of blood lost, on the part of the parents or attendants, who, in estimating the quantity, frequently overlook the fact that the child may have bled into a vessel previously containing water.

Color, Character.—In healthy children free from organic disease the hemorrhage is always arterial, which may be recognized by its bright-red color. The color of the venous hemorrhage, however, which occurs mainly in those suffering from mechanical impediment to the circulation towards the heart, is darker.

Cessation.—Nosebleed is rapidly checked by coagulation in most cases, but if the density of the blood is diminished the coagulation may take place slowly, and a dangerous hemorrhage, attended by prostration, faintness, delirium, and cardiac weakness, may result in consequence.

Treatment.—From what has been stated in the preceding sections, it will be readily concluded that there is greatly more responsibility in deciding when to interfere in many cases of rhinorrhagia than in the selection of any special method of treatment.

In the vast majority of uncomplicated cases in children, spontaneous hæmostasis takes place, and, even when the bleeding is profuse or long continued, mild measures will often suffice to check or keep it within the limits of safety. It is the writer's conviction, founded on experience, that great injury has resulted from the adoption of hasty and heroic treatment in certain cases of epistaxis, and he ventures to suggest the *sero sed serio* régime in appropriate patients.

The treatment consists of rest, and of local, constitutional, and surgical measures.

Rest.—Complete rest of the body is of the first importance, and the child should be placed in a sitting posture, with the head inclined slightly forward as if writing, and not fixed so as to compress the jugular veins. This position of the head places the floor of the nostrils in a horizontal plane, and prevents the flow of blood into the pharynx. The mind of the patient should be quieted, and all fears or excitement abated. The patient should breathe through the mouth; the alæ nasi should be compressed, and all attempts at expelling clots prohibited.

Local and Constitutional Measures.—A compress saturated with cold or iced water may be applied to the forehead of the child, to the dorsum of the nose, or to the nape of the neck. The application of cold or warm bodies to the cervical region, the classic key, and the hot-water bag of Chapman are useful. Voillemier employs a cloth moistened with ether to produce refrigeration over the dorsum of the nose, whilst others make use of cold applications to the scrotum. The application of a strong mustard plaster to the epigastrium or upon the calves of the leg will sometimes be found to arrest the hemorrhage in children.

Snuffing ice-water into the nose, or holding small pieces of ice about the size of a pea in the nostril by means of a tampon, occasionally suffices to check a mild epistaxis. The hemorrhage will often be promptly arrested by introducing into the nose a strong solution of tannic acid (ʒii to ℥iv), or by applying the powder by means of an insufflator. Among the other vegetable astringents may be mentioned kino and catechu, as valuable agents in arresting epistaxis. Fabricius also made use of a styptic powder of his own to arrest nosebleed. Ellis recommends a decoction of matico. Ergot applied locally is said to have checked a profuse epistaxis; but, as it is a painful application, a solution of ergotin applied by means of small pieces of charpie is to be preferred, as it produces no pain. A solution of cocaine (two to five per cent.) either sprayed into the nose or applied by means of small pledgets of cotton introduced gently into the nostril will frequently check an obstinate hemorrhage, but, as already stated, its use will sometimes give rise to a secondary hemorrhage from the diseased area, which may be controlled only with difficulty. Still, it may be tried in severe cases, bearing this fact in mind. When these milder measures fail, the nasal cavity should be treated with iodoform, and then thoroughly plugged with strips of gauze saturated with tannic acid, as recommended by Ingals. These efforts will nearly always be successful, and therefore plugging the posterior nares, which is sometimes a dangerous procedure, as well as a difficult one, particularly in children, should be resorted to only as a last and extreme measure.

A solution of the perchloride of iron is one of the most valuable styptics at our command for the treatment of this affection. The nose should first be thoroughly cleansed of blood by the injection of water, after which a solution of the perchloride of iron (ʒii to ℥ii) should be sprayed into the nose. Vogel recommends a tampon of charpie dipped into a solution of the same substance. Next in importance is the liquor ferri subsulphatis, applied either in the form of a weak spray or by means of a tampon saturated with the solution. Other astringents in solution, such as the acetate of lead, alum, tannic acid, and gallic acid, may also be tried with advantage.

According to Ringer, small doses of the tincture of aconite frequently administered will often arrest nosebleed in children. Turpentine administered in the same manner is also recommended; but the effect should be carefully watched. The same may be said of ergot, which in the hands of some observers has proved beneficial. The hypodermic injection of ergotin has been recommended, but it is liable to be followed by abscesses.

The internal administration of the tincture of the chloride of iron and of the tincture or extract of belladonna and bromide of potassium is highly extolled by some writers.

In the so-called periodic, habitual, or intermittent rhinorrhagias, where malarial influence is suspected, antiperiodics, such as quinine and arsenic, should be administered. Quinine will also be found to have a happy effect, in moderate doses, in many cases in which no history of impaludism can be detected.

Surgical Measures.—Galen recommended compression of the nostrils in mild cases of nosebleed, and their plugging by means of a sponge in obstinate cases. The time-honored expedient of making firm pressure upon the nostril, or upon the septum, by compressing the bleeding nostril with the finger, simultaneously elevating the arm of the affected side above the head, will be found a readily applicable and sometimes a most effectual measure. Deligation of the upper and lower extremities should be resorted to in obstinate cases. It is not an easy matter to discover the bleeding spot, but when it can be done the galvano-cautery can be effectively employed to check the hemorrhage. Owen claims never to have met with a case of epistaxis among children severe enough to require plugging of the anterior and posterior nares: it should, however, be resorted to when all other measures fail, and especially in cases in which a hemorrhagic diathesis is suspected or known to exist.

FOREIGN BODIES IN THE NOSE.

By D. BRYSON DELAVAN, M.D.

Definition.—Foreign substances lodged in the nose, generally by access through the nostrils, but rarely penetrating the integuments from without or passing upward from the pharynx.

History.—The literature of foreign bodies in the nose consisted almost entirely of scattered cases until systematically treated of by Sir Morell Mackenzie in his work on diseases of the nose. While several instances are recorded where such objects as a fragment of shell, a musket-bullet, or the breech-pin of a gun have been forced into the nasal cavity from without, and have remained there undiscovered for a number of months or even years, these accidents have always happened in the case of adults, and do not come, therefore, within the scope of this article.

Etiology.—In dealing with this subject it is necessary to have a clear understanding of the topography of the nasal chambers, and of the nature of the soft tissues with which they are lined. The nasal fossæ are essentially canals, wider at the bottom than at the top, and most spacious through those parts known as the inferior meatus and the middle meatus. Communicating with them are several sinuses, the most accessible of which are the frontal sinus and the antrum of Highmore.

Foreign bodies are likely to be found in that part of the nasal canal possessing the greatest diameter. Hence the inferior meatus is their most common place of lodgement. Again, any object sufficiently small, and capable of locomotion, such as an insect, may find its way into one of the adjacent sinuses.

The mucous membrane which lines the nose is particularly delicate in its construction and acute in its sensibility. It is also highly vascular and capable of an extraordinary degree of distention. A foreign body may, therefore, give rise to great irritation, as may also the attempts of the surgeon at its extraction.

The variety of foreign bodies which have been found in the nose is very great, the list comprising (1) extraneous substances of almost every kind which are of a size to be introduced into the nostrils, either by accident or by design; (2) sequestra of diseased bone which may have come away in the course of necrotic destruction of parts of the bony framework of the nose; and (3) parasites.

The clinical history of the case most commonly seen in practice is as follows. A child about two years of age, old enough to creep, but not yet intelligent, thrusts some small, rounded object, such as a bean or a shoe-button, which it has found in playing upon the floor, into its nostril, and thence into its nasal cavity. If the child be not caught in the act, the body is likely to escape detection, and, the patient being too young to recognize the gravity of the situation or even to describe what it has done, the occurrence is soon forgotten by it and thus passes unnoticed. Soon symptoms of chronic inflammation are established, and the child is thought to have catarrh, thus continuing until finally the actual cause of the trouble is recognized and relieved. Not infrequently, however, the presence of a foreign body exists unsuspected for years, and the child is passed from one physician to another, only to be treated indefinitely for simple catarrh. The writer has seen cases in which a foreign body impacted in the nose had been carried for nine, eleven, and in one instance for fourteen years, although in every instance the patient had been repeatedly under medical treatment.

In a case in which the presence of a foreign body has not been recognized, the first symptom which should call attention to it is the existence of a persistent so-called catarrhal inflammation, confined to one side of the nose. This is apt to be attended with a more or less profuse and fetid discharge, and, while never entirely ceasing, is greatly aggravated by the presence of any of the acute conditions which are usually attended with coryza. Occlusion of the nares of the affected side may not be absolute, although there is generally a decided impairment of the normal breathing-space.

Upon examination by means of anterior rhinoscopy, the unaffected side will appear normal and pervious. In the opposite side, however, will be revealed a condition varying with the duration of the case and the severity of the reaction caused by the irritation arising from the presence of the offending substance.

In cases of recent occurrence the mucous membrane will usually appear congested and swollen, to such an extent sometimes as to conceal the foreign body. In a majority of instances, however, the latter may be seen, lodged in the inferior meatus just posterior to the vestibule of the nose. Where the foreign body has been in this position for a considerable length of time, where its surface is uneven and it has exerted pressure upon the adjacent mucous membrane, the latter will be found more or less eroded, and, in cases of long standing accompanied with excessive local irritation, profuse granulations may exist which so occlude the affected region and surround the foreign body as completely to hide it from view. It is in such cases as these that mistakes in diagnosis are most often made, the patient being told either that the affection is "cancerous," or that he is suffering from syphilitic necrosis, or from lupus,—suspicions which possibly may seem to be confirmed by slight appearances of swelling and redness on the outside of the nose.

Against any such error there is one infallible method of examination,—that, namely, by means of a simple probe, which, passed into the nasal cavity and caused to impinge upon the supposed foreign body, will usually demonstrate in an instant to the sensation of touch the fact that the latter is of abnormal consistency. In rare instances, however, the foreign body has become so covered with inspissated organic matter and viscid muco-pus that the sensation imparted to the probe is that of a soft tissue. In these cases it will generally be possible by slight pressure to vary the position of the body sufficiently to prove that it is an unattached mass. Posterior rhinoscopy, while of occasional service, is of far less value as a means of diagnosis in these cases than the above simple means. It should always be used, however, when practicable, as an adjunct to the anterior method of examination.

In all cases where, after the above manipulation, the presence of a foreign body has been proved or is still suspected, the region anterior to it should be thoroughly cleansed by spraying or douching it with a tepid mild alkaline solution, used until all crusts have been removed and a clear view is obtainable. Following this the application of a solution of cocaine to the mucous lining of the nose in the vicinity of and anterior to the foreign body will generally be found of the greatest assistance in the further treatment of the case, for not only can the nature and position of the substance be studied with greater accuracy, but its removal will also thus be greatly facilitated.

Treatment.—In the treatment of these cases it must be remembered that the operator is dealing with a membrane (1) highly sensitive and often in a condition of hyperæsthesia, (2) distinctly erectile in its nature, and (3) of unusual vascularity. In all cases, therefore, the greatest care should be exercised in handling the parts, lest undue pain be excited. Again, the turgescence of the tissues adjacent to the foreign body renders its extraction all the more difficult by reason of the mechanical obstruction which it offers, while with the slightest irritation bleeding is apt to be provoked.

Should cocaine not be used, and two or three carefully-directed efforts fail to dislodge the foreign body, it is best to place the child under the influence of chloroform, under which the operation can be successfully continued. With the use of cocaine matters are often much simplified. In simple cases a gentle stream of tepid alkaline water, carried in through the unaffected nostril and allowed to flow out through the occluded one, will frequently succeed in extruding the foreign body. A sternutatory has sometimes proved effective. Generally the use of some instrument will be necessary, the choice of which must depend to some extent upon the nature of the foreign body. A small probe with the end bent in the shape of a hook, or a properly-shaped forceps, will be found to answer the purpose in most cases. If the object be lodged far backward, care must be taken in removing it not to push it into the pharynx and thus possibly allow it to fall into the larynx.

Dr. Sajous, of Philadelphia, suggests the following ingenious device. In cases where it is difficult to grasp the object, pass a loop of fine wire through the nostril and *below* the body into the pharynx; then pass another similar wire *above* the object. Draw both loops forward to the mouth, and attach to each a piece of tape. Draw the tape from behind forward until the object is included by it, when the latter may be drawn from the nostril as by a similar device a cork is drawn from a bottle.

After the removal of the foreign body its site is usually marked by an apparently severe condition of ulceration of the mucous membrane. This appearance is in most cases deceptive. The nostril should be washed several times a day with a weak disinfectant, preferably a solution of the permanganate of potassium. In four or five days the membrane will have healed so completely that often no trace of trouble can be seen; the discharge ceases entirely, and cure is complete.

Foreign bodies or inspissated mucus sometimes become the nuclei of the so-called rhinoliths, or nasal calculi, concretions formed by an accumulation of the earthy salts of the nasal secretions. Their presence has not infrequently given rise to so much irritation that the appearances presented have been mistaken for cancer. The history of the case, together with a careful examination with speculum and probe, will easily establish the diagnosis. If the concretion be too large to be easily removed, it should first be crushed by a lithotrite of proper size. Sequestra of bone, particularly in tertiary syphilis, sometimes remain in the nasal cavity after their separation, thus acting as foreign bodies. They must be thoroughly removed, not only as a preliminary measure to further local treatment, but also because, like any other loose object, they may fall into the larynx, with disastrous results. A case is recorded in which a large section of the vomer escaped during sleep and was drawn into the trachea.

In tropical countries, seldom elsewhere, various kinds of flies, of the order *muscidæ*, may enter the nasal cavity, preferably of a patient suffering from catarrh, and there deposit their eggs.

This distressing condition, although by no means confined to that country, has been met with most frequently in India, where the Hindoo practitioners have given it the name "Peenash." Cases are occasionally reported from our Southern States and from Mexico, while even Massachusetts and Illinois have not escaped. The history is usually as follows. While the individual is sleeping in the open air, a fly enters his nose, and, penetrating to a greater or less depth, sometimes even into the frontal sinus, there deposits numerous eggs. These, by the warmth and moisture of their surroundings, are quickly hatched, causing in succession irritability, tickling, and sneezing; later, formication, bloody discharges, and epistaxis, with redness of the face, eyelids, and palate, erysipelas, excruciating pain,—generally frontal,—insomnia, and, if the condition continue unrelieved, convulsions, coma, and death.

Sometimes the larvæ are sneezed out, or they may be seen on examination of the parts, which, of course, will establish the diagnosis.

The destruction caused by them may extend to the mucous membrane, the cartilages, and even to the bones of the head, the ethmoid, sphenoid, and palate bones having been found carious. The extension of the destructive processes is often very rapid. From the terrible nature of the difficulty, as briefly sketched above, it is evident that the true condition of affairs should be recognized at once, and that the treatment should be pursued with great promptness and efficiency. The old method consisted in syringing out the offending objects by the aid of various mixtures, prominent among which were solutions of alum, of tobacco, and of chamomile, while insufflations of calomel and numerous sternutatories were also used. That these means were entirely inefficient is plain when the impossibility of causing fluids to penetrate the sinuses, and the unfortunate results of the cases so treated, are considered.

More rational and scientific is the method proposed by Dr. John Ellis Blake, and first published by him in *The Boston Medical and Surgical Journal*, April 10, 1862. This consists in the application to the orifice of the sinus of the vapor of chloroform or of ether, preferably the former. The effect of this is to cause the maggots to seek relief from suffocation by escaping with all haste to the outer air, so that they are not only destroyed, but also at the same time removed. This latter is a most important feature, for by the old plan, even if the solution used succeeded in destroying their activity, they still remained behind to irritate the parts as foreign bodies and to become probable sources of septic infection. In some cases the simple inhalation of the anæsthetic has been effective; in others, however, a more thorough application seems to have been required, and for these the injection into the nasal cavity of chloroform diluted with water has been recommended.

We would deprecate this latter procedure as both painful and dangerous, and would again urge the value of the drug cocaine, suggesting that it be used as follows: first gently cleanse the nasal space with a mild alkaline solution; then secure complete insensibility and complete retraction of the mucous membrane by means of cocaine, applying it with especial care to the vicinity of the canal leading to the frontal sinus, should that cavity seem to be invaded, in order that the approach to it may become as patent as possible; finally, allow the chloroform to be inhaled, and, if this do not succeed, place some chloroform in an atomizer and force it into the upper and anterior part of the nasal space in the form of spray.

Extreme cases have been reported where, post mortem, the larvæ were found so deeply embedded in the tissues that they seemed beyond the reach of drugs, no matter how applied.

Besides the larvæ above described, cases, fortunately very rare, are recorded, in which leeches, ascarides, earwigs, and centipedes have been found in the nose, where their presence has caused insomnia, frontal pain, sanious discharge, lachrymation, vomiting, and, in some cases, great cerebral excitement. Sternutatories have generally succeeded in effecting their expulsion. It may be necessary, in extreme cases, to trephine the frontal sinus.

In the management of all cases of foreign body, of whatsoever nature, it is plain from what has been said that early recognition of the condition is of the utmost necessity, for not only will much pain, trouble, and expense be spared the patient, but consequences most serious and even fatal may be averted. Careful and thorough examination, therefore, of the nasal cavities cannot be too urgently insisted upon.

TUMORS OF THE NOSE.

By D. BRYSON DELAVAN, M.D.

Neoplasms of the nose may be benign or malignant. They may be situated in the nasal fossæ proper, or they may originate from some point in the naso-pharynx. The former region is rarely attacked in children, while the latter during adolescence is a not infrequent site of the growth known as the naso-pharyngeal fibroma. Of new growths of the nose it may be said, in general, that the varieties usually found in other parts of the body may occur in the nasal fossæ. Some forms, however, are so rare as to be little more than pathological curiosities in the adult, and in earlier life they are unknown. Of benign growths the variety most commonly met with in the adult, the simple myxoma or so-called mucous polyp, is in the child rare. Occasionally adenomata, angeiomata or erectile tumors, enchondromata, and osteomata may occur. There have been found also growths composed of some of the elements of a dermoid cyst, and, somewhat analogous to these formations, a hitherto nameless variety presenting a structure similar in its elements and in their arrangement to the helix of the ear.

NASAL POLYPI—MUCOUS POLYPI—MYXOMATA.

These tumors are defined by Sir Morell Mackenzie as a new formation, generally of the nature of a myxoma, but sometimes containing a small amount of fibro-cellular tissue. They are usually pedunculated, round, oval, or pyriform in shape, of a pale pinkish color, semi-transparent, varying in size, and causing in the nasal passages more or less obstruction, with its associated symptoms.

That the presence of polypi is due to such influences as heredity or struma has not been proved. That they may arise from mechanical irritation is more probable, although the actual causes are unknown. The affection appears to be more common among males than among females. The youngest case seen by the writer was that of a boy aged thirteen, and one case is on record in which it occurred in a boy of twelve. Of two hundred cases reported by Sir Morell Mackenzie, but sixteen were under twenty years of age.

The earliest symptoms of this condition are such as would attend an ordinary coryza. There is, generally, marked susceptibility to taking cold.

The attacks of coryza increase in frequency and in severity. Owing to the hygroscopic quality of the polypoid tissue, the growths tend to increase in size during an attack of coryza and in wet weather, so that at such times they cause a greater degree of occlusion than when in their ordinary condition. Later, headache is a common symptom, and this is occasionally associated with disturbance of the mental processes. If left to themselves, their growth continues until complete occlusion of the affected nasal passage results. The discharge is of an ordinary and simple character. Epistaxis sometimes occurs. The olfactory sense is either blunted or destroyed, by reason of the mechanical obstacle to the entrance of the inspired air and with it of the odorous particles to the olfactory region. Taste is also more or less impaired, while the nasal canal and the Eustachian tube may be obstructed. The growths most commonly originate from the middle turbinated body, and next in frequency from the neighborhood of the superior turbinated body and superior meatus. The septum is rarely the site of the affection. Mucous polypi are usually multiple, although it sometimes happens that a single large growth will be found. When, however, one large tumor has been removed, it will almost always be possible to discover others still existing. They are usually pedunculated, but may also be found of rounder shape, with broad base and entire absence of pedicle. Zuckerkandl believes that the pear-shaped variety arise from sharp edges, while those with broad bases occur upon flat surfaces. The surface of the growths is smooth and shiny, and when touched they are felt to be elastic. They themselves are devoid of sensibility, although the mucous membrane in their neighborhood may be in a highly sensitive state.

Pathologically, the covering of mucous polypi is composed of ciliated epithelium, beneath which are generally a few dilated capillaries, but no nerves. The bulk of the growth is made up of embryonic connective tissue, consisting of a hyaline gelatinous material through which more resisting cellular trabeculæ pass in various directions. The gelatinous substance is very rich in mucin, and contains in its early state round and oval cells, which at a later period become elongated, fusiform, or stellate, and for the most part nucleated and granular. The latter kind of cell is said to be the most common. The consistency of the growth depends upon the greater or less degree in which the connective-tissue stroma or the mucous substance predominates in its structure. Here and there small cavities, full of a colorless, stringy fluid, may be met with which are regarded by some as true cysts. Again, true cysts have been observed in the neighborhood of polypoid growths.

The presence of mucous polypi is seldom attended with danger, although they may give rise to serious local and reflex irritation.

Recently the reflex influences of nasal polypi have been extensively studied. Their presence may be followed by well-marked nervous phenomena. Thus, asthma has often been cured by their removal, while reflex cough, hemicrania, supraorbital neuralgia, vertigo, and even epilepsy, have been attributed to them, and chronic coryza and hay-fever are often due to their presence.

Mucous polypi show a decided tendency to recur, owing probably to lack of thoroughness of removal, and also to the probable presence of smaller growths, which, being relieved from the pressure of their larger companions, increase rapidly in size. Again, the tendency seems especially marked in certain individuals.

Diagnosis.—The diagnosis of mucous polypi of the nose is generally easy. Their appearance is characteristic, while their softness, elasticity, and freedom from pain render them unlike any other growth. They also differ from growths of a more serious nature in that they do not bleed when touched and rarely cause disfigurement. Deviations of the septum can hardly be mistaken for them if both sides of the septum be carefully examined, nor can hypertrophy of the mucous membrane of the septum if cocaine be applied to it. Abscess of the septum and other growths of this part of the nose can be differentiated from polypi by the appearances present, and by the fact that polypi so rarely spring from this locality. The greatest difficulty likely to occur is in distinguishing polypi from hypertrophy of the mucous membrane covering the middle or lower turbinated bodies. Here the easiest mode of differentiation is by the application of cocaine, under the influence of which hypertrophied mucous membrane will at once become retracted, while when the drug is applied to the polypi no apparent effect is produced. By means of a small probe, delicately handled and used in connection with anterior rhinoscopy, the consistency of the growth may be determined and its attachments accurately studied.

Treatment.—The surgical measures for the removal of nasal polypi are three in number,—viz., evulsion, abscission, and the galvano-cautery. Of these, evulsion with the forceps is by far the oldest and the one most generally practised. The success which attends its employment will be due entirely to the operator's knowledge of the anatomy of the nasal chambers in general, and of the precise location of the growths to be attacked. The old-fashioned way of setting blindly to work with a pair of polypus-forceps and tearing away whatever might come into the grasp of the instrument, without knowing what tissue has been seized, cannot be too strongly reprehended. The result of such careless operating is, necessarily, the infliction of severe and unnecessary pain, the production of copious hemorrhage, the possible serious injury of healthy parts, and the imperfect removal of the growths. In all cases the polyp should be removed by the aid of the rhinoscopic speculum or mirror, care being taken not to work in the dark. No one instrument is likely to meet the requirements of every case, so that a variety of forceps is desirable. In using the forceps the pedicle of the growth should be grasped and the polyp removed as near to the mucous membrane as possible. It is well not to attempt to remove too many growths at one sitting, for, although the operation may be accomplished with comparative thoroughness at one time, it is impossible to do wholly satisfactory work when the parts are obscured by blood. Working by the aid of a good light, with the growths clearly in view, it is comparatively easy to detect their attachments and thereby save the patient much pain.

The application of cocaine to the nasal mucous membrane preliminary to these operations is of the greatest value, not only in rendering it non-sensitive, but also in causing it to retract and thus bringing the growths into plainer view.

For the abscission of nasal polypi the best instrument at present in use is a small-sized, light Jarvis écraseur, carrying a loop of the finest-sized piano-wire. With this the pedunculated growths may be removed easily, without hemorrhage, and with a minimum of pain. It is particularly useful in the case of growths originating in the superior and posterior parts of the nasal cavities.

Removal should be followed by some plan of treatment calculated to destroy thoroughly any remaining polypoid tissue. Applications to the site of the polyp of strong solutions of iodine, chromic acid, or carbolic acid may be used for this purpose. The galvano-cautery itself affords an excellent means for the removal of polypoid growths, and in some respects is superior to any other method. It causes, on the whole, less pain, and is preferred by many patients. Several applications, however, are usually required to secure the complete removal of the myxomatous tissue. Something may be accomplished in preventing the development of nasal polypi by relieving the chronic inflammations of the nasal mucous membrane which are favorable to their growth.

Prognosis.—The prognosis as to recurrence is often uncertain. The possibility of a simple myxoma degenerating into a growth of a more malignant character, while denied by Billroth, has of late received confirmatory evidence in the hands of Michel, Hofmann, Schmiegelow, Schaeffer, and Bayer. The latter reports a case in which a carcinomatous area was discovered within a large mucous polypus. Cases are not wanting, too, in which after the repeated removal of polypi the nasal mucous membrane has assumed a condition of irritation which, to say the least, is slow to subside, and which renders it a fertile soil for the production of new growths of a less benign nature.

ADENOMATA.

These growths, which are very rare, consist in an hypertrophy of the glands of the mucous membrane. They would be harmless but for their tendency to undergo epitheliomatous degeneration. The tumor first bears a resemblance to a mucous polyp: it is, however, of firmer consistency, approaching somewhat that of an enchondroma. Its progress is slow, so long as degeneration has not begun; when this becomes established, the course is that of an ordinary epithelioma. In the period of transition the diagnosis can be made only by the aid of the microscope. The treatment must be surgical.

ANGEIOMATA.

Angeiomatous growths of the nose are rare. A careful study of the subject was made by Dr. John O. Roe and published in the "Transactions of the American Laryngological Association" for 1885. By including in his list all growths in which the vascular element predominated,—such as erectile tumors or nævi, vascular tumors, angeiomata cavernosa, and the fibro-angeiomata,—there were found the records of but thirteen cases, and to these Dr. Roe has added one. In analyzing them it was found that in two instances the tumors were attached to the cartilaginous septum, while the others, as far as the origin was designated, grew from the upper parts of the nasal fossæ, as, for instance, from the vomer, the basilar apophysis, the inferior surface of the body of the sphenoid, and the vault. In but two cases were the middle turbinated bodies involved. In but one instance was the growth located in the right naris, while in no case has it been reported as growing in both, either alternately or simultaneously. Of the fourteen cases but two were women. The youngest case was thirteen, and in eight cases the patient was under twenty. The duration is uncertain, several cases having suffered from epistaxis since childhood, while in one case a tumor of unusual proportions developed within six weeks.

Of the symptoms recorded, epistaxis is by far the most common and well marked. Indeed, it was present in every case, and the attacks were generally profuse and persistent.

Five of the fourteen patients died, four as the direct result of operation. Of these, one was operated on by Nélaton's method, one by resection of the superior maxilla, one by removal with a curette through an opening made in the side of the nose, and one by an attempted removal with forceps. Four cases are now recorded in which removal was effected by means of Jarvis's cold-wire écraseur. The success attending this method is most gratifying. In operating with it great deliberation may be used, and thus even less hemorrhage be produced than with the galvano-caustic loop, so that the procedure may be regarded as entirely safe. Its superiority over the surgical measures employed in the four cases mentioned above is obvious. In using the cold wire it will be desirable to have a galvano-cautery at hand, so that, should the removal of the tumor be only partial and hemorrhage ensue, the remnant of the growth may be thoroughly obliterated, and thus the best prospect for the relief of the bleeding secured.

OSTEOMATA.

These are bony tumors of the nose, rare, and most often encountered in the young. That they are the result of any diathesis is improbable. The causative influence of traumatism is more apparent, particularly in the injuries to the nose so common to young children. Specific treatment has no effect in checking their progress. Sex seems to make no difference.

Pathologically, the structure of these growths does not differ from that of osteomata in general. In the nose they are generally of dense structure, but sometimes cancellous, and they are not connected with the bony framework of the nasal cavity. Their size varies greatly, and they may attain large dimensions, and thus cause serious destruction to the neighboring parts.

The early symptoms are obscure, and the patient seldom seeks relief until the tumor has attained considerable size. Those which may be present at first, however, are coryza, epistaxis, and a marked sensation of itching.

As the tumor enlarges, there is more or less obstruction to nasal respiration, loss of the olfactory sense, and often neuralgic pain of a severe character, due, no doubt, to pressure upon nerve-filaments and becoming worse as the growth increases in size. Anterior rhinoscopic examination reveals a tumor covered with mucous membrane, rose-red, dark red, or even purplish in color. Later there is ulceration, and even necrosis may be present. There is usually at this time a fetid discharge, resembling that from syphilitic disease of the nose. Meanwhile, the parts upon which the growth encroaches may be eroded, or else forced from their normal positions, and thus distinct deformity, external as well as internal, may be produced. Neuralgia may give place to anæsthesia as the irritated nerves become more forcibly compressed. Left to themselves, nasal osteomata increase in size until they fill the nasal fossæ, encroach upon the pharynx, the maxillary sinus, the orbital cavities, and the base of the skull, and finally give rise to grave cerebral complications. In other cases they may cause serious symptoms from caries, necrosis, and suppuration. They are usually recognized and removed before these accidents have taken place, and show little tendency to recur after extirpation.

Prognosis.—The prognosis is generally good, provided the growth can be removed through the natural passages.

Diagnosis.—In the earlier stages the diagnosis may be somewhat difficult, the growth being readily mistaken for an exostosis or a nasal calculus. Osteomata, during the early part of their course, are somewhat movable, while an exostosis is not. A rhinolith is of a more friable consistency, as may be demonstrated by exploring its surface with a long needle or a fine probe. Enlargement of the turbinated bodies, particularly the middle turbinated, might be mistaken by an inexperienced observer. The tissues covering the turbinated bodies, however, are soft, lax, and yielding, and quite unlike the tense investment of an osteoma. No other nasal neoplasm is likely to cause so much neuralgia, except cancer, while the growth of the latter is far more rapid. Fibrous tumors of the nose are very rare.

Treatment.—The only treatment is extirpation. The cancellous osteoma can be crushed with strong forceps and removed in fragments. In the case of ivory-like growths it has generally been considered necessary to lay open the nose, preferably, if possible, by Rouge's operation, which consists in separating the upper lip and base of the nose from the superior maxillary bone and reflecting them upward over the face, so that the anterior nares are brought into immediate view. Should this not give sufficient space, some other and more radical surgical procedure is advised. To the great credit of American surgery be it said, in this country such unnecessary and dangerous methods have been superseded by the use of the burr of the dental engine, as first suggested by Dr. D. H. Goodwillie, of New York City. By means of this instrument, in skilful hands, the most difficult cases have been successfully treated, the entire operation being accomplished through the natural passages, with a minimum of shock to the patient, with none of the after-dangers of a capital operation, and with absolutely no disfigurement to the external parts.

ENCHONDROMATA.

Cartilaginous tumors of the nose are so rare that hardly a dozen cases are on record. This form of new growth is incident to youth, all cases thus far observed having been under eighteen years of age. In a case seen by the writer the patient was but two and a half years old.

The symptoms in general are those of nasal obstruction. The tumor is never pedunculated. It usually springs from the cartilaginous septum, or, rarely, from the outer wall or roof of the nose. The prognosis is good, and there can be little excuse for removing the growth by the way of any but the natural passages. The diagnosis may be made from the position of the growth, its method of implantation, its consistency, which is readily determined by means of a fine exploring needle, and its insensibility. It need never be mistaken for a deflection of the septum, since when the latter exists there is a corresponding depression upon the opposite side. Removal in most instances may be accomplished successfully by means of the knife, the cold snare, or the galvano-caustic loop.

EXOSTOSES.

Exostoses of the nasal fossæ, other than those occurring low down upon the septum in the shape of bony ridges or spurs, must be very rare. The treatment of the latter will be described in another connection.

DERMOID OR EMBRYONAL CYSTS.

Of the nature of these growths are certain abnormalities which have occasionally been observed in the nasal fossæ. They may consist partly or altogether of hair, or they may contain cartilage, bone, fat, connective tissue, and glandular substance. The presence of teeth in the nose has been observed in several instances, and seems to be the most common accident of this class.

Several cases of osseous cyst of the nasal fossæ have been reported. These growths are apt to originate from the septum or the inferior turbinated bone. Their symptoms are analogous to those of an osteoma, and their progress is slow, but progressive. Treatment consists either in the radical extirpation of the cyst or in incision and destruction of it by the aid of the galvano-cautery. If the cyst be multilocular, each division must be opened in turn, and this treatment continued until the growth has disappeared.

MALIGNANT TUMORS.

Malignant tumors of the nasal fossæ, of primary origin, are rare. With the exception of sarcomata, they occur more commonly after middle life. Sarcomata, however, have been found in very young children. Of the cases seen by the writer in the past fifteen years a majority have been in males, although the experience of others has been to the contrary. Sarcomatous growths are apt to develop rapidly and to extend to the various cavities in their vicinity, causing much destruction to the surrounding parts, and consequent deformity.

The symptoms generally present are obstruction of the affected side, followed later by epistaxis and the secretion of a sanious, fetid, muco-purulent discharge. Respiration is, of course, impeded, the quality of the voice is changed, and the olfactory sense is impaired. Neuralgia is a marked and distressing symptom. It may attack one or both sides of the head, and it generally increases in severity until the nerves pressed upon by the growth are paralyzed. Upon examination the tumor, in the earlier stages, is seen to be simply red and perhaps nodulated; later it becomes ulcerated, grayish in color, and covered with an unhealthy secretion. It bleeds at the slightest touch.

As to the diagnosis of malignant growths of the nose in general, their early recognition is often extremely difficult. It is also most important, for upon it will depend, in great measure, the prognosis. The question of syphilis, although not easily eliminated by inspection, may be settled by studying the results of specific treatment. Lupus is nearly always attended with external manifestations of that disease. Great rapidity of growth, especially of a tumor which has recurred after removal, is characteristic of sarcoma. Microscopical examination is nearly always possible, and should always be made.

The prognosis, as in the case of cancerous disease in other parts of the body, is grave. Sarcomata seem to be the least malignant in their nature, and with them the prognosis is not absolutely bad.

The older authorities agree that the only proper method for the treatment of malignant growths of the nose is thorough and radical extirpation, and they generally recommend that in order to render the operation a success the growth should be fully presented to the operator by means of a preliminary operation. This, however, with the improved methods and instruments at present used for operations upon the nasal cavities, is, in a large number of cases at least, by no means necessary. Thanks to the efforts of Voltolini abroad, and of Dr. R. P. Lincoln, of New York, the value of the galvano-caustic and electrolytic methods for the cure of these cases has been abundantly proved. Hypodermic injections of a thirty-per-cent. solution of lactic acid into the substance of the tumor have seemed to cause an arrest of the growth, while the persistent application to its surface of astringent solutions is also sometimes palliative. Attacks of hemorrhage may usually be controlled by the use of the galvano-cautery.

TUMORS OF THE NASO-PHARYNX.

Fibrous Tumors, or the so-called naso-pharyngeal fibromata, are of fibrous structure, and generally originate from the vault of the pharynx, whence they may extend in various directions, causing absorption or destruction of the neighboring parts and giving rise to much annoyance and danger to the patient. The disease is rare. Dr. R. P. Lincoln, of New York, has succeeded, however, in tabulating fifty-three cases. Of these, thirty-eight were genuine fibromata. All occurred in males under twenty-five.

This disease is incident to youth, and is almost unknown among females. Some believe that it may be caused by scrofula or by bad hygienic surroundings. Much more plausible is the explanation of Sir Morell Mackenzie, who believes it due to the irregular evolution, during the growing period, of a tissue which under normal conditions is exceptionally abundant on the under surface of the base of the skull. It seems possible that it is to an exaggerated plastic activity during the period of most active growth that these tumors owe their origin.

The early symptoms are those of obstruction to the nares and of the presence of an unusual object in the pharynx. Obstruction to respiration increases with the enlargement of the growth, and, in case the tumor extend far downwards, dyspnœa may become severe. Deafness, from pressure upon the orifices of the Eustachian tubes, and loss of the sense of smell, may be present, while articulation becomes thick and indistinct, and occasionally there is severe dysphagia. An abundant purulent secretion, sometimes very fetid, is generally present, while epistaxis is such a common and severe symptom that it may become a prominent and dangerous feature of the case. Marked drowsiness and general debility are often observed.

The appearance of the tumor is usually smooth, its consistence hard and unyielding, and its color red or bluish purple, while its surface is often ulcerated. Its exact seat of implantation seems to be the periosteum covering the basilar process of the occipital bone and the body of the sphenoid. Other apparent points of attachment are merely secondary adhesions, formed in the course of the expansion of the growth. Later in its development the tumor begins to cause deformity of the adjacent bony structures, the nature of which will depend upon the direction taken by the growth. As it advances, everything gives way before it, and even the cavity of the cranium may be invaded. In some instances seen by the writer, prolongations have been found to extend in many directions, almost every free space in the track of the tumor having been encroached upon and the skull penetrated as before mentioned.

The diagnosis is generally not difficult if the tumor have attained considerable size. The age and sex of the patient, the appearance of the growth, and its rarity will usually establish the diagnosis, while any question as to the existence of sarcoma may be settled by the microscope.

The prognosis, unless treatment be begun at an early stage, is unfavorable. There seems to be a tendency to absorption after the age of twenty-five, so that if the disease can be held in check until that time a cure may be effected. Rarely, spontaneous sloughing followed by recovery has taken place.

Treatment.—There will be few cases of this formidable disease in which surgical interference, in some form or another, will not be called for. For its removal two general varieties of procedure have been proposed: first, the old method of removal after the performance of a so-called preliminary operation, by means of which the region invaded by the growth was brought directly within reach of the operator; and, secondly, the new method, by which extirpation is successfully accomplished through the natural passages. When the remoteness of the location of such a tumor and the complicated nature of its ramifications are considered, it will be at once evident that the extent of a preliminary operation may be unlimited, and that under the hand of a bold operator the safety of the patient may be seriously imperilled. That such has often been the case, the enormous rate of mortality attending these operations will abundantly prove. With the means of observation now at our command the region most likely to be invaded can be thoroughly examined by direct inspection, so that the presence of the tumor can be recognized at an early stage and competent means for its relief applied which later might be inexpedient or of less avail.

Of the preliminary operations for gaining access to the naso-pharyngeal cavity three varieties have been recognized,—namely, the nasal, the palatine, and the maxillary. Of late years the method of dealing with these growths has undergone such a change that preliminary operations, although still occasionally performed, may well be relegated to the past. Those interested in them may find them described at length in the older works upon surgery. The modern methods include two principal resources: first, the electric cautery, and, secondly, electrolysis. Both are used through the natural passages.

In the employment of the electric cautery the best plan is, if possible, to surround the base of the growth with the wire of the galvanic écraseur, passed either through the nose or through the mouth, and then to effect its separation by the application to the wire of a moderate degree of heat. Too great incandescence and the too rapid separation of the tumor will be followed by bleeding, while with greater deliberateness and care an almost bloodless removal may be accomplished. Any remnant of the stump which may be left should be thoroughly destroyed by means of the electric cautery, applied at intervals of a week. For this purpose several good cautery electrodes are now to be had.

Electrolysis can be applied by any battery generating a continuous current of moderate strength. One or more curved needles, connected with the negative pole, should be introduced directly into the tumor, while the positive pole meanwhile is applied to the sternum. The operation should continue from ten to fifteen minutes at a sitting, and be repeated every day or two. From this method excellent results have been reported. The extraordinary advantages in these cases of the less heroic plan of treatment have been proved by R. P. Lincoln, both by the record of his own cases and from statistics of twenty-one cases of fibromata in which a preliminary operation was performed. Of these, three patients died upon the table and a fourth succumbed within a few hours, while a fifth nearly died of hemorrhage while the operation was in progress. Of eight cases in which the operation was performed through the natural passages, the patient recovered without accident in every instance. The rate of mortality from the old method would doubtless be greatly increased if the unsuccessful cases had invariably been placed upon record.

Fibro-mucous Polypi, although occasionally seen in the adult, are in the child exceedingly rare. They are not prone to bleed, and show little tendency to return when removed. They may be extirpated by evulsion, although the Jarvis cold-wire écraseur affords probably the easiest and best method for their removal.

Sarcomata.—The symptoms of this growth are similar to those of fibromata, with the addition, in certain cases, of nerve-pain, of a lancinating character, which is apt to be referred to the ear and to be worse at night, severe dysphagia, and general cachexia. The diagnosis must be established by the aid of the microscope. The prognosis is absolutely bad. The progress is rapid, recurrence after removal almost certain, and in many cases there is a disposition towards the formation of secondary deposits in other organs. Early recognition and thorough removal by the galvano-cautery may so modify the prognosis that it is possible that better results than those heretofore attained may be reached.

Dermoid Tumors of the pharynx have been reported in about forty instances. They are evidently congenital, and must be caused by the misplacement, during an early period of fœtal life, of embryonic elements intended for the formation of structures at the opposite and external extremity of the Eustachian canal.

Arnold, in an exhaustive article[1] upon this subject, concludes as to their origin and to their relation with teratoma that those should be considered heterogenic teratoma in which the origin can be traced to the secondary development of already existing embryonic elements, while those are autogenic which originate from the development of abnormal embryonic elements, or from the misplacement or dislocation of abnormal embryonic elements, or from the misplacement or dislocation of normal ones.

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[1] Virch. Archiv für Path. Anat., January 5, 1888.

In a case seen by the writer, a pale, rounded excrescence, about a fourth of an inch thick in its antero-posterior dimension, extended downward from behind the velum palati to a point about half an inch below its free border, and from the left lateral wall of the pharynx to the median line. It was attached to the posterior aspect of the hard palate, immediately below the orifice of the left nasal fossa. Its presence seemed to have caused no particular irritation, and it had remained unrecognized for twenty years. The tumor was covered with a pilose integument, it contained a distinct double plate of cartilage, and the whole structure was identical with that of the helix of the ear.

CONGENITAL SYPHILIS OF THE NOSE.

BY F. H. BOSWORTH, M.D.

SYPHILIS in the father or mother is exceedingly liable to be followed by syphilis in the child. Whether a syphilitic father can transmit syphilis to the offspring without infecting the mother, or whether the mother must first be infected and thereby transmit the disease to the child, is still a matter of discussion among syphilographers. Again, it is still an open question as to how long after the primary lesion the father or mother can transmit the disease. The weight of opinion, however, I think, leans decidedly to the view that the limit of transmission in either father or mother is certainly within three years after the contraction of the disease. These problems, however interesting, need not be entered upon in the present article.

As a result of inherited syphilis, the fœtus in utero may become syphilitic, giving rise to premature birth, or the child may be still-born at the end of the full term; or, again, the manifestation of the disease may be delayed until after birth, although if the disease is present it shows itself very soon. Thus, Von Rosen[1] found that, out of sixty-eight cases, in all but nine the disease manifested itself earlier than three months after birth; Kassowitz,[2] out of one hundred and twenty-four cases of hereditary syphilis, found symptoms presenting in eleven cases in the first week, twenty-one in the second, thirty-four in the third and fourth, forty in the second month, and eighteen in the third month; and Roger,[3] out of two hundred and seventy-two cases of hereditary syphilis, found the symptoms presenting in one hundred and twenty-two cases in the first month, one hundred and twenty-eight cases in the second and third, and only thirty-two later. Baümler[4] comes to much the same conclusion, in making the general statement that nearly half the children are attacked in the first month of life, one-third in the second, about one-eighth in the third, and only one-eleventh at a later period; he adds, however, that the symptoms very seldom begin in the first week, and not infrequently in the second; while Didet[1] makes the statement that he has seen a few cases as late as four months, and one case in which the disease did not appear until the child was nearly two years old. This last statement is somewhat unique, and it is not improbable that this child may have acquired the disease after birth, as it scarcely harmonizes with our knowledge of the action of the syphilitic virus that it should remain latent for two years in the nursling.

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[1] Behrend's Syphilidologie, New Series, iii., 1861, pp. 223 and 226.

[2] Die Vererbung der Syphilis, Vienna, 1876.

[3] Recherches cliniques sur les Maladies de l'Enfance, 1883, vol. ii. p. 1.

[4] Ziemssen's Cyclopædia, vol. iii. p. 240.

The present consideration is confined entirely to the discussion of hereditary syphilis, which is occasionally treated of under the designation of congenital syphilis. Now, the latter term may be used to describe a form of syphilis which is acquired at birth, namely, by infection from an existing lesion in the genital passages of the mother. In this case, I am disposed to think that the disease runs an entirely different course from that to be described in the present article, and probably differing in no very marked degree from the ordinary type of acquired syphilis, although these cases are so exceedingly rare that the data upon which any general statement is based must necessarily be, to a certain extent, somewhat limited: hence, when Simon[2] makes the statement that "congenital syphilis not infrequently manifests itself for the first time after weeks, months, or in certain instances not until the age of puberty and even after many years," I think if by congenital syphilis he means the hereditary form of the disease the statement must be accepted with considerable reservation. If, however, he alludes to syphilis acquired at birth, his assertion may undoubtedly be correct, with reference to the very late manifestation of the disease, although even here there is an element of doubt, in that the syphilis can be acquired in irregular or accidental ways and the primary lesion escape observation; for in cases where the disease is acquired at birth we should usually expect a more rapid course of development than in adult life, although by no means so rapid a course as is met with in the hereditary form of the disease.

The earliest manifestation of congenital syphilis in children is either coryza or some form of cutaneous eruption. I know of no statistics bearing on the frequency of the special lesion, although, unquestionably, in the large majority of cases the first manifestation of syphilis in children occurs in the form of a coryza, which manifests itself by the ordinary symptoms of nasal stenosis, with watery discharge, which as the disease progresses gradually develops into a muco-purulent discharge of a somewhat acrid character, giving rise to irritation of the margins of the nostril and the upper lip, together with crust-formation about the vestibule.

The essential lesion consists of an inflammation of the mucous membrane lining the nose, apparently a non-specific rhinitis. We probably have no means of making a definite diagnosis of the especial lesion in these cases, since not only is the examination of the nasal cavity exceedingly difficult in young children, as revealing any easily recognizable condition, but, moreover, if an examination were feasible, it is questionable if the morbid appearance would afford any special light in directing attention to the existence of syphilis. The diagnosis, therefore, must depend entirely on the clinical history of the case and on the concomitant appearances, mainly on the general appearance of the child, who shows very marked evidence of malnutrition, the skin presenting a pale, somewhat earthy color, while the general facial expression gives to the child a pinched and old-man face, as it were. In connection with this, in the majority of cases, either concomitant with the development of the nasal symptoms or soon after, there appears the ordinary eruption on the skin, making its appearance usually about the anus or buttocks, and afterwards spreading over the body. This is usually papular in character, presenting the typical copper-color. A further manifestation of the disease in the nose consists in the deposit of gummatous material, either in the superficial or the deep layers of the membrane, which, breaking down rapidly, results in an ulcerative process. This phase of the disease is manifested by an increase of the pus-discharge, which has now assumed a somewhat offensive character, mingled with blood and shreds of black necrotic tissue. The secretions from the ulcerative surfaces form hard incrustations, which, drying and piling up by a somewhat rapid process of accretion, attain such size that they cannot be expelled from the cavity, and hence form an additional source of irritation, in that they may give rise to reflex brain-disturbances, which may lead to the suspicion of the existence of some form of brain-syphilis. In most cases, probably, however, this is due simply to the fact that the incrustations accumulate to such an extent as completely to block the passages and prevent the escape of the purulent discharge. Hermann Weber[1] has reported two cases of infantile nasal syphilis in which epileptiform convulsions with coma developed coincidently with the cessation of the sanious discharge from the nose, the nervous symptoms disappearing immediately upon the re-establishment of the escape of pus from the nose. A fair inference here might be that the damming back of the pus gave rise to septic infection; but septicæmia is one of the rarest of complications in nasal syphilis, even where extensive necrosis has resulted from the disease. A true explanation of the development of the brain-symptoms in these cases is to be found in the fact that in young children the retention of the crusts produces reflex disturbances of the nerve-centres.

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[1] Traité de la Syphilis des Nouveaux-nés et des Enfants à la Mamelle, Paris, 1854.

[2] Virchow's Spec. Path. u. Therapie, art. "Syphilis," Band ii., Abth. 2, p. 577.

Congenital syphilis of the nose in young children runs an exceedingly rapid course, the ulceration following rapidly on the coryza, leading to exposure of bone and subsequent necrosis. External deformity shows itself very early in the history of the case, evidencing the fact that the whole of the cartilaginous septum and probably some portion of the vomer or even the nasal bones have been destroyed. In a case reported by Hawkins[1] nasal syphilis developed in a child six weeks after birth, resulting in complete destruction of the vomer, with sinking in of the nose, four months later. We thus find the clinical history of the development of syphilis in children differing from that of adults in a very striking degree. This is not to be explained by the view that inherited syphilis is a more active poison than the acquired form of the disease, but rather by the fact that small children possess a comparatively slight power of resisting the inroads of any disease: hence the syphilitic virus makes a very powerful impression from the onset upon infants, giving rise to a general impairment of all the nutritive powers, as evidenced by their general cachexia already described, this general cachexia not being necessarily a direct but rather an indirect result of the disease.

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[1] Med.-Chir. Trans., vol. xliii. p. 188.

Diagnosis.—The diagnosis of nasal syphilis ought to be comparatively easy in the early stage, where it is characterized by a simple coryza. It should be remembered that the turbinated tissues are in a very early stage of development at birth and for some months later; hence an acute idiopathic rhinitis is an exceedingly rare disease at this age. Furthermore, if by any chance such a disease exist, it would run the ordinary course of a few days and undergo resolution, whereas in syphilis it progresses rapidly towards the development of a discharge of such a decidedly purulent character as to eliminate the possibility of its being an acute rhinitis, even in its late stages, wherein the discharge never obtains an absolutely purulent character. In a purulent rhinitis in children, in the commencing stage of atrophy, the disease never develops earlier than from three to four years of age, and at its onset is an exceedingly mild affection, and not characterized by any notable stenosis or great swelling of the mucous membrane. Hence, in a given case of coryza in the first three months of life, if in any degree persistent, suspicion should always be excited of the existence of inherited disease. If, on the other hand, the child is small, ill nourished, and presents the ordinary appearance of anæmia, together with an earthy tint of the skin, and an old-man look in the face, we have still further confirmation of this suspicion. The appearance of the characteristic eruption, however, renders the diagnosis complete, and should be easily recognized from its gross appearances. According to Baümler,[2] this usually presents the typical copper-colored, elevated papules present on the buttocks or about the anus, which very soon assume the appearances of mucous patches. In still rarer cases the eruption may be of the macular character, although Romberg[3] and Van Harlingen[4] assert that the smooth macular eruption is more frequent. In either case, however, the minute extravasation is characteristic of syphilis, as shown by the copper-colored tint of the eruption. Still later the discharge of bloody pus mixed with necrotic tissue, in connection with the characteristic fetor which attends an ulcerative process in the nose, of course leaves no possibility of mistake in diagnosis. Baümler[1] alludes to the characteristic appearance of the external nose in these cases, which consists mainly in a depressing or flattening of the nasal ridge, together with protrusion of the frontal sinuses. This feature is oftentimes noticeable, and is not to be regarded as an evidence of any destruction of the nasal bones as the result of disease, but is rather due to the fact that in consequence of the stenosis the alæ of the nose are sunken in, and the nostrils to a certain degree collapsed. As a result, the bridge of the nose presents a somewhat bulbous or swollen appearance, which is heightened by the emaciation of the child, the features being pinched, as it were, and, the subcutaneous cellular tissue being absorbed, the thin flabby skin drawn firmly across the nose gives it a misshapen aspect, which, as before stated, is only an appearance, and not an absolute condition.

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[1] Contributions to Pathology and Surgery, vol. i. p. 229.

[2] Loc. cit., p. 238.

[3] Klin. Ergebnisse, Berlin, 1846, S. 178.

[4] International Encyclopædia of Surgery, vol. ii. p. 453.

In addition to these subjective symptoms, much light can also be thrown on the matter of diagnosis by making close inquiry as to the possibility of syphilitic disease in either the father or mother. A patient may oftentimes attempt to deceive a physician where the inquiry is made in regard to acquired syphilis in his own person. When, however, a man is confronted with the possibility of having transmitted a loathsome disease to his offspring, he is usually disposed to answer questions with absolute candor and honesty. Hence this clinical feature of the disease can usually be investigated very thoroughly, and the facts of the case established with a fair degree of certainty. The same considerations, I take it, apply with equal force to the mother, who in a matter of this kind will confide the truth to the physician, even if she have something to conceal from the husband. As a matter of clinical observation, any father or mother who has had a primary syphilitic lesion within three years preceding conception is liable to transmit that disease to the offspring. Whether this possibility of transmission occurs later, is still an open question. Clinical facts, however, I think are rather against it, and hence our investigations should be made on this basis. Still another interesting question is as to the possibility of the father transmitting syphilis to his child without first infecting the mother. Clinical facts are about equally distributed in supporting one or the other side of this question, and yet, on purely physiological grounds, it is not easy to understand why this may not be,—especially as the possibility of inheriting the rheumatic, gouty, and tubercular diatheses from the father remains unquestioned.

Prognosis.—The early development of syphilis in children is to be regarded as the evidence of the activity of the specific virus in the system. Thus, in a case where the evidences of the disease are present at birth, the prognosis is simply bad, as those cases are rarely amenable to treatment,—one of the most serious features of the case being that the nasal disorder so far interferes with nursing as to lead to the very early development of malnutrition or marasmus, and the children usually succumb, largely as a result of this complication. On the other hand, we may state it as a rule that the later the development of the disease the more favorable the prognosis, in that the child has had an opportunity of gaining vigor and strength to combat the blood-poison when it manifests itself; and, furthermore, I think it may be safely asserted that the later the disease manifests itself the slower its progress, and hence the better the opportunity for establishing the diagnosis and placing the child under proper remedial measures. In the ordinary run of cases—namely, in those cases in which the coryza or syphilis sets in from four to six weeks after birth—the prognosis is based largely on the general condition of the child, many children showing at this age very marked evidence of marasmus. In these cases the prognosis is bad. If, on the other hand, we have to do with a fairly well nourished child at four to six weeks of age who develops syphilitic coryza, when we consider the fact that we possess a specific remedy in mercury and that children come readily under the influence of this drug, the prognosis may be considered favorable, if the disease is recognized and the remedies administered sufficiently early. If ulceration, with resultant necrosis, has developed before the disease is recognized, this does not in itself modify the prognosis as regards ultimate recovery, other things being equal. In other words, if a syphilitic child has developed necrosis at six months without treatment and still shows no very marked evidence of general malnutrition, there is no reason for giving an unfavorable prognosis, in that the syphilitic explosion, as it were, which leads to the deposit of gummatous material in the mucous membrane lining the nose, seems to have exhausted itself in this deposit, and the further progress of the disease is largely a local process, in that the mass breaks down into an open ulcer, under which all the gummy material which formed the original deposit is thrown off. The necrosis of bone which results entails a much longer process for its exfoliation. This process, however, does not necessarily lead to any impairment of the general health, except so far as it interferes with the normal function of the nose, interfering with nursing and thereby producing impaired nutrition. If what has now become a local disease acts to impair the general health, it acts indirectly and not directly. As we have already seen, syphilitic disease of the nose in children runs an exceedingly rapid course, but still adheres to the same rules which govern the manifestations of syphilis in the adult. It shows a marked hesitancy in transgressing anatomical boundaries, and does not extend, therefore, to the integument anteriorly nor into the pharynx behind. If it produces destruction of the hard palate, this is due to the original deposit of gummy material in the bones forming this structure, or, if the ethmoid or sphenoid bones are involved, this involvement is due to original syphilitic disease. In those cases, of course, where the extent of involvement of tissue in a necrotic process is very great, the prognosis must necessarily be, to a certain extent, rendered more grave, in that the general health must suffer in a young child in whom so large an extent of ulcerative action is going on. We come, then, to the conclusion that a fairly correct prognosis can be made at the time the disease is recognized, and is based on the time at which the disease develops, the extent of tissue involved, and, lastly, but of most importance, on the general condition of the child.

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[1] Loc. cit., p. 239.

Treatment.—The local treatment of the coryza is a matter of some importance, if thereby we are enabled to restore the passages to their normal patency and thus allow the child to take its nourishment in proper amounts from the breast. For this purpose we perhaps possess no other remedy so efficacious as cocaine, which should be used in the form of a spray in about one-half per cent. solution, as follows,—

℞ Cocaine, gr. iii ;
Boracis, gr. vi ;
Aquæ ad ℥i ;

or, perhaps better still, in the form of an emulsion with some oily substance, such as the following :

℞ Cocaine, gr. iii ;
Aquæ, ♏ x.
M., ft. sol., et adde
Ol. menth. pip., ♏ v ;
Ol. amygdalæ, ℥i.
S.—Shake before using.

Either of the above can be used with some simple hand-ball atomizer, and may be placed in the hands of the attendant to be applied to each cavity every two or three hours. Astringents possess no value in this condition. The integument about the margins of the nostrils is always exceedingly tender, and should be protected by the local application of vaseline or cold cream. A certain amount of good is undoubtedly done in these cases by anointing the external nose either with mutton tallow or, better still, with the well-known domestic remedy, the tallow of a goose.

When the disease has progressed to the ulcerative stage, our efforts are directed entirely towards keeping the parts thoroughly cleansed, by means of a wash, after the membrane has been reduced and the passages opened as far as possible by one of the cocaine solutions already mentioned. For the wash any simple alkaline lotion may be used. The difficulty, of course, in cleansing the nose in an infant is that the child cannot blow its own nose. This is fairly well accomplished for the child by fitting the nozzle of the spray-apparatus into the nostril and pressing the bulb, the reservoir of the spray being empty ; the theory being that if you blow into one nostril of the child the palate is immediately thrown up against the posterior wall of the pharynx and that orifice closed, and hence the current of air escapes with considerable force through the other, carrying with it such mucus or pus as may lie in the passages. If this is not successful, there is no serious objection to using a cotton pledget firmly fixed on the end of a probe, as after the use of cocaine the parts are by no means sensitive. If ulceration has set in, the efforts should be towards controlling this, in that the disease runs an exceedingly rapid course, and bony necrosis must necessarily occur unless the ulcerative process is arrested, for we are justified in believing that a gummy deposit does not always extend deeply into the mucous membrane, and hence necrosis is not always an inevitable result of an ulcerative action. Our best method of controlling ulcerative action is by the use of iodol or iodoform. This should be applied, after the parts are thoroughly cleansed, in the form of a powder, by means of insufflation. Neither of these drugs is irritating, and hence they can be used in full strength.

By far the most important treatment of nasal syphilis is to bring the child as rapidly as possible under the influence of mercury, the administration of which must be regulated by much the same rules as govern its administration in adults. If for any reason the administration by the mouth cannot be well managed, inunctions answer an excellent purpose, the amount of mercurial ointment to be used being about five grains daily, or, still better, the oleate may be used, two or three minims daily of a twenty-per-cent. strength. Ordinarily, however, I think the internal administration of mercury is preferable, and for this purpose we may use either mercury with chalk or calomel in doses of one grain twice or three times daily, or protiodide in doses of one-eighth or one-tenth of a grain. In children, as in adults, the administration of the protiodide is liable to cause disturbance of the bowels. This, however, can be regulated by the administration of a small quantity of opium. If for any reason we are obliged to abandon internal medication, we must resort to inunctions or mercurial baths. The mercurial bath may be prepared by dissolving eight or ten grains of corrosive sublimate in four or five gallons of tepid water, into which the child is placed and allowed to remain from ten to fifteen minutes, care being taken to exclude water from the eyes, mouth, and nose. If ulceration exists in the nasal cavity, or evidence of gummy deposit, it is well to administer small doses of iodide of potassium for a limited period of time, this period being governed by the toleration of the child and the impression which the remedy makes upon this special feature of the disease. The dose, however, cannot ordinarily be increased above, possibly, two grains given three times daily. In this case it is probably wiser to confine the administration of mercury to either the biniodide or the bichloride, as in this manner any danger is avoided of forming in the system a poisonous combination of the iodine with mercury, although this objection to the combined administration of these two remedies has probably been over-estimated. In addition to the constitutional treatment, the general condition of the patient ordinarily demands the administration of tonics, and especially the use of cod-liver oil, while at the same time the most scrupulous care must be exercised in the observation of the utmost cleanliness in the child's surroundings, by the daily administration of the cold bath, and by subjecting the child to the best of general hygienic influences.

ACUTE CORYZA.

By CARL SEILER, M.D.

Definition.—There is perhaps no affection more common in infancy and early childhood than acute coryza, or cold in the head, or sniffles as it is commonly called by mothers and nurses; and, because it is so common and apparently trifling an affection, little attention is paid to it by those who have the care of children. And yet, trifling as it seems in most instances, it is a disease which should by no means be neglected or made light of, for serious consequences may result. The disease may be defined as an acute inflammation of the mucous membrane and the underlying erectile tissue of the nasal cavities, characterized by swelling of the tissues, heat, and dryness, followed by a more or less copious discharge of thick mucus or muco-pus, which by its presence, together with the swelling, produces more or less complete obstruction of the nasal cavities and thus prevents nasal respiration.

As is well known, the nose in infants and small children is relatively smaller than the other features of the face, and the anatomical relation of the parts within the nasal cavities is slightly different from that in the adult. Thus, Kohts and Lorent have observed that the meatuses in the child's nose are very narrow, and that the lower turbinated bone projects farther into the nasal chamber than it does in the adult, thus making the breathing-space very small indeed. In the same way, the middle turbinated bone is also curved more towards the septum, and is frequently cleft so that apparently it consists of two wings, so to speak, while the upper portion of the anterior nasal chambers is filled up by the superior turbinated bone, which may also be cleft. This anatomical arrangement does not allow of much enlargement of the soft parts by swelling, and, consequently, even a slight tumefaction of the mucous membrane or a slight thickening of the normal nasal secretion will produce obstruction of the nose. As the child grows older the turbinated bones are gradually drawn away from the septum, thus increasing the size of the meatuses; and, consequently, an attack of coryza does not produce as disastrous effects in older children as it does in infants who have not as yet learned to breathe through the mouth with comparative comfort.

Symptoms.—An attack of coryza is usually ushered in with more or less severe general febrile disturbances. The first symptoms which call attention to the nasal mucous membrane as being the source of disturbance are sneezing, congestion of the conjunctiva with increased flow of tears, a slight watery discharge from the nostrils, and obstruction to nasal respiration. The infant at the breast will grasp the nipple in its mouth to suck, but will let go of it almost immediately to catch its breath, thus being unable to obtain the necessary amount of nourishment, and the pangs of hunger are added to the other symptoms. This catching of the breath through the mouth often resembles an attack of laryngismus stridulus, and may be mistaken for acute laryngitis. In older children this, of course, does not occur, yet even they often experience great difficulty in eating or drinking.

In the course of a few hours a watery discharge is seen to issue from the nostrils, which on coming in contact with the skin produces a tickling sensation, and the child, in order to relieve itself, sniffs it back again, and therefore the name "sniffles" has been given to the affection. This discharge, containing as it does almost all the salts of the blood, is acrid and irritating, so that it causes a soreness of the skin of the upper lip and the edges of the nostrils unless the child is kept scrupulously clean.

In the course of a few days the discharge becomes thick and yellowish in color, and the obstruction to nasal respiration is thereby increased, and the pharynx and larynx become involved in the inflammatory process in consequence of the enforced mouth-breathing. The febrile symptoms which usually disappear with the appearance of the watery discharge again make their appearance, and older children complain of headache and loss of appetite, and are restless during sleep and listless and disinclined to play during the day. A slight hacking cough, with gagging and even vomiting, particularly in the morning, is noticed in many cases, due to the accumulation of the thickened nasal secretion in the naso-pharyngeal cavity. Tinnitus aurium and dulness of hearing, with occasional earache, are also noticed in some cases where the inflammation has extended to the openings of the Eustachian tubes, and this may lead to middle-ear catarrh and perforation of the tympanic membrane.

The conjunctiva also becomes affected in many cases, being injected; and watering of the eyes, with a thickening of the secretion, closing the lids together during sleep, is observed.

As a rule, in the ordinary non-infectious cases the discharge becomes gradually less, until, in the course of a week or ten days, it has entirely disappeared and complete health has been restored.

In cases, however, in which the coryza is due to necrosis of the bones in the nasal cavity, or to foreign bodies introduced into the nose, or if it is of gonorrhœal origin, the discharge keeps up indefinitely, and may become purulent and sanguineous. If it is due to the presence of nasal polypi,—which, however, is very rarely the case in young children,—bleeding from the nose is quite frequent, while in the ordinary forms of coryza nosebleeding is seldom met with.

In one variety, which is infectious but not contagious, and is usually seen in connection with acute pharyngitis and tonsillitis, the febrile symptoms are absent at the onset of the disease, and do not, as a rule, develop until the second or third day, while the attack is usually ushered in by some muscular pain and great weakness.

Etiology.—The cause of acute coryza in the majority of cases is a chilling of the surface of the body or of the feet by getting them wet. Insufficient clothing, leaving the child long without changing a soiled or wet diaper, insufficient drying of the skin after a bath, may bring on an attack. Kicking off the bedclothes at night, undue exposure to wet or cold during the day, and insufficient ventilation in the bedchamber are also causes of coryza in older children. A number of cases of coryza in infants have been observed which were caused by undue exposure of the eyes to strong light, the irritation thus started in the conjunctiva extending to the nasal mucous membrane either by direct infection through the tear-duct or, more likely, by reflex nerve-action.

Many authors assert that a frequent cause of coryza in new-born children is the introduction into the nose of the secretions of the maternal vagina; but, unless these secretions are of a specific gonorrhœal nature, it is very unlikely that they will give rise to a coryza in the child. It is a well-known fact that the unbroken mucous membrane of the nasal cavities does not readily absorb any infectious material, and even surgical procedures in children and adults can be carried on without antiseptic precautions without producing septic wounds or even suppuration, because the abundant secretion of mucus so covers the surface of the mucous membrane that infection seems impossible. On the other hand, a systemic infection is a very frequent cause of acute coryza, and thus it is a common symptom of most of the zymotic diseases of childhood, while at the same time the systemic poison may find its expression in an inflammation of the upper air-passages only, as is the case in influenza and in the lately-observed form of infectious pharyngitis without initial febrile symptoms.

Congenital syphilis is also a frequent cause of coryza in infants, and is distinguished in nothing from the ordinary acute disease except in the persistence of the symptoms when the cause is not recognized and the treatment is not anti-syphilitic. In older children suffering from syphilitic coryza the discharge is frequently bloody, and emits a disagreeable odor, due to the ulcerative process having attacked the bony structures, thus causing necrosis.

Gastric and intestinal irritation caused by indigestion or the presence of worms in the bowels is another frequent etiological factor in the production of the disease. Finally, the presence in the anterior nasal chambers of foreign bodies, such as pieces of paper, cherry-stones, shoe-buttons, small pebbles, and other like articles, which children are very apt to push up their nostrils without being noticed by their parents or guardians, gives rise to an attack of coryza which, like that of syphilitic origin, becomes prolonged, and the discharge sanguineous and fetid, unless the foreign body is removed.

Polypi and neoplasms are rarely met with in the nasal cavities of infants and young children, while in older children they are not infrequent and by their presence give rise to all the symptoms of acute coryza.

Method of Examination.—The treatment must first of all be directed to discover and remove the cause of the disease, and, consequently, a careful inspection of the anterior nasal chambers of the child is necessary.

The method best adapted for this purpose, in the writer's experience, is to insert a small rubber ear-speculum or delicate nasal dilator into the nostril, and reflect a strong beam of light from a window or lamp, by means of a concave head-reflector which is fastened to the examiner's head by a head-band, in the same manner as it is used in laryngoscopy. If the patient is an infant, the examination is best made while the child is lying in its nurse's arms and its head held steady by the nurse's hands. With older children it often becomes necessary to confine the arms and legs, which is most easily accomplished by enveloping the little patient in a sheet folded lengthwise, so that it can be wrapped around the body several times, confining both arms and legs. The head should be tilted slightly backward, and the source of light which the head-reflector reflects should be above and behind the patient's head. If old enough to understand the importance of the procedure of the examiner, it is best not to restrain the child at all, but to proceed as gently as possible with the necessary steps of the examination, letting the little patient handle and examine every instrument to be used, thus gaining his confidence, when, with a little expenditure of time and a good deal cf patience, the physician will be able to examine the anterior and often even the posterior nasal chambers of a child in precisely the same manner as that adopted for adults.

In this way the anterior nasal cavities can be inspected, and the condition of the mucous membrane and the turbinated tissue, as well as the presence of ulceration, foreign bodies, and neoplasms, can be determined by inspection. But it often becomes necessary to insert a probe to determine with accuracy the precise character of a swelling, neoplasm, or foreign body, and this should be done with great caution, for, if the child is once frightened by being hurt during an examination of this kind, it will rarely submit to another without a great deal of struggling and crying. The writer has found it best, therefore, to apply to the mucous membrane a four-per-cent solution of cocaine on a pledget of absorbent cotton, before using any exploratory instrument in the nose of children. Cocaine solutions, in whatever strength, should never be used as a spray in the nasal cavities, because such an application does not sufficiently localize the anæsthesia to the spot which it is desirable to render insensible, and because it is apt to pass into the post-nasal cavity and from thence into the stomach in such quantities as to give rise to toxic symptoms.

After the cocaine has been allowed to remain for five or ten minutes in contact with the spot to be examined, the pledget of cotton is removed, and the examination with the probe can be carried on gently without giving rise to any pain or annoyance to the little patient.

A posterior rhinoscopic examination in young children with the rhinoscopic mirror is impossible in the majority of cases, and even in older children is attended with great difficulty. Should the symptoms, however, point towards an obstruction of the post-nasal space, the index finger of the examiner can easily be inserted behind the velum palati and the nasopharyngeal cavity explored by the sense of touch.

An examination thus conducted will reveal the mucous membrane intensely congested, and the turbinated tissue swollen so as to touch the septum and thus almost completely obliterate the breathing-space. During the first stage the mucous membrane appears dry and slimy, while in the latter stages it is covered by the more or less thick nasal mucus, which must first be removed by spraying or by mopping with moist absorbent cotton before its surface can be seen. Any ulceration, neoplasms, or foreign bodies will then show themselves to the eye and to the touch of the probe.

It is very essential to inquire carefully into the previous history of the case, particularly with new-born infants, so as to be able to arrive at a correct idea of the cause of the disease and institute the proper treatment at once should it prove to be a case of gonorrheal or syphilitic coryza.

Treatment.—Very little is to be said about the treatment of this disease, because in most varieties the cure depends upon the removal of the cause and upon the good judgment of the attending physician. The febrile symptoms should be combated in the usual way with aconite and spiritus ætheris nitrosi in small often-repeated doses graduated to the age of the child, and a small dose of calomel and soda as a mild purge.

The patient should be kept as quiet as possible, in a warm but well-ventilated room; and, if it is an infant at the breast, feeding with a spoon should be at once commenced, so as to supply the nourishment which it cannot obtain in the usual manner. In the case of older children the nostrils should be cleansed three to four times a day with a spray from an atomizer containing a solution made according to the following formula:

℞ Sodii bicarb., ʒ viii;
Sodii bibor., ʒ viii;
Sodii benzoat.,
Sodii salicylat., āā, gr. xx;
Eucalyptol.,
Thymol., āā, gr. x;
Menthol., gr. v;
Ol. gaultheriæ, gtt. vi;
Glycerini, ℥ viiiss;
Alcoholis, ℥ ii;
Aquæ, q. s. ad Oxvi.

With infants and young children this cannot easily be done, but a swab of absorbent cotton dipped in the solution can be used to cleanse the nasal mucous membrane. This gives great relief to the little sufferers, and amply pays for the trouble which it occasions to the nurse.

The external rim of the nostrils as well as the skin of the upper lip should be well anointed with cacao butter or cosmoline, and even the skin covering the nose itself should be kept well greased.

If the cause of the disease is of an infectious nature, change of air, if such is possible, is of great benefit, and, if the coryza is the result of the infectious pharyngitis lately observed, small doses of benzoate of sodium (one-fifth of a grain) every hour will speedily break it up.

Plenty of cold water should be allowed, and in older children the diet should be chiefly milk and bread.

Powders of an astringent nature, such as tannic acid, nitrate of silver, sulphate of zinc, and others, should *never* be used as local applications in the anterior or posterior nasal cavities of children; nor should astringent solutions be used in the form of washes or sprays, as they invariably give rise to increased swelling of the turbinated tissue and thus make the little patient worse rather than diminish the discomfort.

As a local disinfectant in those cases in which the disease is due to the presence of micro-organisms, a spray of Labarraque's solution diluted in the proportion of one part to four of water, or a spray of peroxide of hydrogen diluted (1 to 5), thrown into the anterior nares, after they have been cleansed with the antiseptic solution for which the formula is given above, will aid materially in checking the flow of mucus and diminishing the swelling of the turbinated tissue.

If the coryza is merely a symptom of one of the zymotic diseases of childhood, the cleansing of the anterior nares gives great relief and aids materially in keeping the fever down, and in many cases prevents the distress and suffering occasioned by a dry tongue and throat, because these latter conditions are largely due to the enforced mouth-breathing.

In those cases in which a careful inspection of the nasal cavities reveals ulceration, which in children is invariably due to congenital syphilis, it is best not to wait for the slow action of internal or even local medication, but surgical measures should at once be instituted to remove all dead bone and necrosed tissue with a small nasal curette. Very little if any hemorrhage will follow such an operation, and much permanent damage and disfigurement of the face can be prevented by such timely surgical interference.

If foreign bodies are detected, they should be removed at once; and one of the best instruments for the purpose is Gross's ear-spoon and hook.

Polypi or other neoplasms should also be removed at once by means of the Jarvis snare, an instrument which, if properly used, gives rise to little bleeding and pain, and is far superior to the now old-fashioned and barbarous polypus-forceps formerly used for the purpose.

Although these surgical procedures can readily be carried out with little or no pain by anæsthetizing the parts with cocaine solution, yet it is better to administer a general anæsthetic to the little sufferer, for only when unconscious will a young child submit to a lengthy operation within the nasal cavities.

After all obstructions by foreign bodies or neoplasms have been removed, the after-treatment should consist simply of frequent cleansing of the nasal cavities with the alkaline antiseptic solution, and in a short time the normal nasal respiration and secretion will return and the troublesome disease will have disappeared.

RHINITIS HYPERTROPHICA.

By WILLIAM CHAPMAN JARVIS, M.D.

Synonyme.—Hypertrophic nasal catarrh.

Definition.—A chronic inflammatory affection of the nasal passages, characterized by an abnormal increase or hypertrophy of the pituitary membrane and permanent dilatation of the blood-sacs of the turbinated structures.

Etiology.—By far the most common local cause of nasal catarrh in children, infants excepted, is distortion of the nasal septum. Inherited asymmetry and narrowing of the nasal fossæ will frequently be discovered in these cases of deformity of the septum. Catarrhal inflammation of the pharyngeal tonsil may develop a chronic rhinitis. Traumatism, foreign bodies, and polypi act as local causes of the complaint. Interference with the escape of venous blood through the spheno-palatine foramen produces congestion of the pituitary membrane (Spicer). Idiopatic causes of rhinitis hypertrophica are undue exposure to cold, exposure to draughts, wetting of the body and especially of the feet, excessive humidity, and constant breathing of a dusty atmosphere. Insufficient food, impaired nutrition, general cachexia, syphilis, and scrofula favor the occurrence of the disease. Repeated attacks of acute rhinitis may finally give rise to a true hypertrophic process.

Pathology.—While an increase in the thickness of the entire mucous investment of the nasal chambers is discoverable in hypertrophic rhinitis, the unique feature found in this affection, and in no other disease of mucous membranes, is a series of changes in the so-called turbinated bodies. These erectile structures exhibit increase of the epithelial layer with round-celled infiltration, thickening of the submucous cellular tissue, excessive formation of connective tissue, and dilatation of the cavernous sinuses with paresis of their contractile walls (Jarvis).[1] Posterior turbinated hypertrophies may be either soft and dark or hard and pinkish white (Seiler). The pituitary membrane is congested throughout the entire nostril, and presents hypertrophy of the epithelial and subepithelial structures. Pathological enlargement of the tonsillar and adenoid structures in the vault of the pharynx is commonly associated with the rhinitis hypertrophica of childhood.

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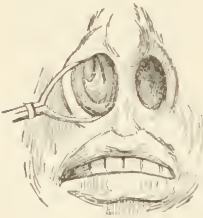
[1] Medical Record, May 27, 1882.

Complications.—In addition to the common result of the long-standing hypertrophic processes, nasal stenosis, there may exist an obstructive enlargement of the adenoid and tonsillar tissue in the vault of the pharynx. Proceeding from this point downward, one may meet with pharyngitis (follicular or catarrhal), laryngitis, tracheitis, bronchitis, and bronchitic asthma,—these secondary affections being the result either of impaired respiration, of accumulation of secretions, or of extension of inflammatory processes.

Organs more remotely connected with the nasal cavities may be likewise involved, as the ear, in the form of a purulent otitis, and deafness may result from interference with the pneumatic patency of the Eustachian tubes, or the eye, in the shape of a conjunctivitis from inflammatory or obstructive implication of the lachrymal duct and sac. The accessory nasal sinuses and cavities may be occasionally included in the catarrhal processes. Finally, the general health of the child may be impaired, through interference with nutrition and normal respiration, and the imperfect performance of the last-named function may result in a preternatural narrowing of the thorax.

Diagnosis.—The determination of the existence of hypertrophic rhinitis is usually accomplished without much difficulty, since the pathological features of the disease are, as a rule, well marked. Anterior rhinoscopy reveals the presence of localized and general redundancies of the intra-nasal mucous membrane, abnormal increase in the size of the turbinated tissues, chronic engorgement of all the intra-nasal structures, and narrowing of the diameter of the normal dark outlines of the nasal respiratory slit, occasioned by the encroachment of a deflected septum or hypertrophied turbinated body. Fig. 1, taken from a life-sketch, exhibits a bilobate, antero-inferior turbinated hypertrophy in the nostril of a child (female) twelve years old.

Fig. 1.



Posterior rhinoscopy reveals the presence of large glandular masses in the vault of the pharynx, concomitants of nasal catarrh, and hypertrophy of the posterior portions of the inferior turbinated bodies.

The symptomatic signs are chronic rhinorrhœa in infants, and inability of sucklings to take continuous nourishment, attacks of suffocative spasm from obstructed respiration, habitual mouth-breathing, constant sneezing; and in children frequent complaint of headache and earache, nasal cough, constant raising and expectoration of ropy mucus, inability to breathe through the nose, especially during the night, with consequent disturbance of rest, dryness of the throat, and mental inaptitude.

Prognosis.—In regard to the life of the individual, a direct fatal issue is possible only during infancy. At this period it may occur in nurslings from insufficient nourishment (Rayer). Nasal stenosis, by robbing an infant of its rest, may ultimately induce exhaustion (Frank), or may lead to the development of pulmonary hyperæmia (Kussmaul). After the period of infancy the prognosis is in every respect excellent. The radical relief of the leading symptom, nasal stenosis, is promptly followed by free nasal drainage, the easy expulsion of intra-nasal accumulations by means of the respiratory blast, recuperation of the pharynx, larynx, and lungs by the cure of the enforced mouth-breathing habit, improvement in hearing, through restoration of the pneumatic aural equilibrium, and removal of a train of dependent eye-symptoms. As a natural result, a great improvement in the general health of the child soon becomes apparent. The respiratory tract is placed in the most favorable state for its ultimate and expansive development, and the impulse given to the nutritive processes is followed by a corresponding change for the better in the physical and mental well-being of the child.

Treatment.—Childhood is *par excellence* the period most favorable for the radical treatment of catarrhal disease of the nose. The prompt employment of rational therapeutic measures at this time is, by reason of the incipiency of the morbid processes, obviously more likely to effect a cure than later in life, when the affection has become well stamped. Furthermore, many of the sequelæ and complications of catarrhal disease may actually be prevented or avoided by the early employment of active therapeutic measures.

Treatment, both surgical and therapeutic, has principally for its object the removal or correction of two prominent conditions,—namely, nasal stenosis and the excessive formation and accumulation of intra-nasal mucus.

The methods commonly employed to overcome nasal obstruction are *reduction* and *excision*.

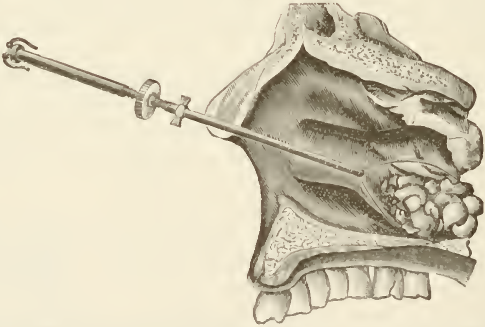
Hypertrophy of the turbinated tissues and deflection of the septum are most frequently responsible for nasal stenosis, and consequently require this treatment. The turbinated tissues may be reduced by means of cocaine, the action of the drug in this respect being, however, only temporary. These structures and the deflected septum may likewise be reduced by the application of the galvano-cautery. While the galvano-cautery as a reducing agent possesses the advantage of permanency in its effect, it is not entirely free from certain objections attending its use. This means, as generally applied in the form of the incandescent platinum point, accomplishes the desired object through the extraordinary cicatricial contraction incident to the healing of the furrows burned into the obstructing turbinated tissues. The common forms of electric apparatus employed for this purpose are the plunge battery and the storage cell. Both accomplish this object in a satisfactory manner, the preference lying mainly in the matter of cell-construction. The two most convenient forms of plunge batteries known to me are those of Dr. Seiler, made by Fleming, of Philadelphia, and Dr. Robinson's, sold by Stammers, of New York. After a diversified experience with many varieties of apparatus, my preference has been finally exercised in favor of Gibson's (of New York) storage cell, as affording one of the most powerful, manageable, compact, portable, and reliable means for electrical supply now found in the market.

The other forms of reducing agents—injections of ergotin, galvanization, elastic pressure, nasal sounds, etc.—may be safely dismissed with a mere mention, since their general utility and effectiveness are as yet largely matters of conjecture.

The other method employed to overcome nasal stenosis—namely, that of excision or removal of the offending tissues—is by far the most valuable, by reason of the simplicity and effectiveness of the means at our disposal. The most important of these are the snare, destructive action of caustics, ligature, excision (scissors), ablation (tearing), and disintegrating injections.

The cold-wire loop, when properly employed, offers the simplest, safest, and most effective means for the removal of redundant turbinated tissues and, occasionally, obstructive distortions of the septum. Fig. 2 shows the écraseur devised by me to remove vascular turbinated hypertrophies with facility and with little or no pain and hemorrhage. A detailed description of the manner in which the instrument is employed may be found in special articles.[1] Anterior turbinated hypertrophies are removed by simply pressing the wire loop against the loose redundant structure and slowly pinching it off. Occasionally the hypertrophy will be found to be too firm to permit of its being seized in this manner. In these cases it will be

FIG. 2.



The above illustration, taken from Dr. Lefferts's work on "Chronic Nasal Catarrh," conveys an excellent idea of the position assumed by the écraseur when adjusted for the removal of posterior turbinated hypertrophies.

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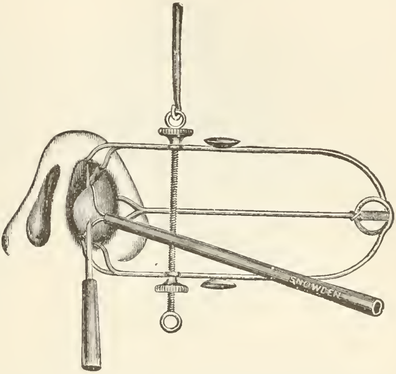
[1] Dr. F. Bosworth, "Jarvis's Operation, its Relation to Nasal Catarrh," Medical Record, July 9, 1881; Dr. Carl Seiler, "Jarvis's Operation in Hypertrophic Nasal Catarrh," Medical Record, October 29, 1881; Dr. F. I. Knight, Medical News, January 21, 1882.

necessary to employ my transfixion-needle. The delicate needle is simply thrust through the base of the hypertrophy, the wire loop being then carried over the point and heel of this device. (Fig. 3.)

FIG. 3.



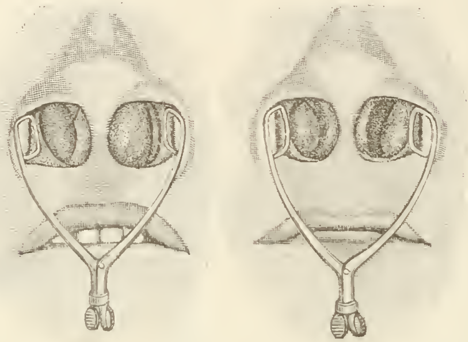
TRANSFIXION-NEEDLE AND SNARE, adjusted for the performance of the operation. (From Dr. Sajous's work on "Diseases of the Nose and Throat," the author having kindly furnished me with the illustration.)

Turbinated hypertrophies may also be removed by the persistent application of escharotics. The most popular agents of this kind are chromic acid, monochloracetic acid, nitrate of silver, and nitric acid. Chromic acid is applied upon a flattened copper probe, the affected tissues being first bathed with a solution of cocaine. Nitrate of silver may be applied first upon a probe or in the form of the mitigated stick. Nitric acid may be applied upon a glass rod or by moistening the point of a splinter of wood with the destructive fluid. Acetic acid is recommended by Dr. Bosworth, of New York, being employed by him in the same manner as explained for chromic acid.

Localized cartilaginous deviations of the septum may be readily removed by means of the snare and transfixion-needle. Extensive deflections, osseous or osseo-cartilaginous in character, require the use of special cutting devices. Adams's fracture-forceps, Steele's stellate punch, Bosworth's hand-saw, Roe's electric saw, Seiler's gouges, and many forms of nasal burrs have been employed, more or less effectively, for this purpose. The electric drill furnishes a speedy, effective, and manageable means for removing intra-nasal distortions. These I have had constructed of steel tubing, to facilitate antiseptic cleansing. The C. and C. electric motor propelled by a single storage cell furnishes ample power for running the antiseptic tubular drills. Cocaine is, of course, employed. The importance of the surgical treatment of the malformed or deflected septum in children is more apt to be overlooked than to be overestimated, for it has been determined that these distortions are more readily corrected at this time of life, when the nostrils are plastic and undeveloped. Certain it is that the golden opportunity to nip a catarrhal process in the bud, which if left untreated may end in life-long discomfort, should not be lightly dismissed. (Fig. 4.)

The therapeutic management of chronic rhinitis, while valuable in childhood, is especially indicated in infancy. Therapeutic measures are employed to free the nasal passages from accumulations of mucus, for the healing of catarrhal excoriations, the softening of nasal incrustations, the lubrication and palliation of the inflamed mucous membrane, and the cure of hemorrhagic abrasions.

FIG. 4.



HAZARD, HAZARD & CO. W. F. FORD.

ANTERIOR DEFLECTION OF THE NASAL SEPTUM, before and after its removal by means of the antiseptic tubular drill. (From a life-sketch.)

Detergents and antiseptic washes are employed to carry out the first indication. They relieve the stenosis by softening and washing away the intra-nasal incrustations and thickened mucus. The devices usually employed to render these applications effective are the anterior and posterior nasal syringe, several forms of nasal douches, and various spraying devices. The post-nasal syringe offers a most thorough means for flushing the nasal cavities. When organic stenosis exists, removable, however, by surgical measures, ear-trouble may be caused by fluids being forced into the Eustachian tube. The utility of this means is lessened or lost with infants and very young children. In this class of cases some form of anterior nasal douche or spray may be employed. A simple and useful douche of this kind, extemporized by myself, can be had by fitting the nasal nozzle of a Politzer air-bag to the rubber bulb of a Warner douche. A peculiar pitcher devised by Politzer to pour fluids through the nose may be also utilized. Thudichum's or Weber's gravity douches are also sometimes, but less satisfactorily, employed. A variety of substances have entered into the composition of nasal lotions, among which may be mentioned bicarbonate, biborate, benzoate, phosphate, and chlorate of sodium, in the proportion of from one to five grains to the ounce of fluid. These bland, unirritating salts of sodium are usually employed in solution with glycerin and a mere trace of an antiseptic agent, like peppermint, menthol, salicylic acid, benzoic acid, carbolic acid, or bichloride of mercury. Nasal lotions are, of course, always warmed before using.

Astringents are of doubtful efficacy as applied to the pituitary membrane. It is possible that they may occasionally act as mild local tonics.

These solutions should be very weak. Ferric alum and sulphate of zinc (gr. ss–i to the ounce) and glycerite of tannin (gtt. x to the ounce) are most commonly employed in the form of a spray.

Powders, on account of their irritating qualities, are contra-indicated in hypertrophic rhinitis.

Unguents are sometimes useful to soften and soothe the inflamed mucous membrane and to prevent the formation of intra-nasal incrustations. They may be sprayed into the nose or applied by means of a brush. Sprays of heated vaseline prove very grateful.

These local measures will naturally have to be combined with appropriate treatment of the effects and complications of the catarrhal processes, commonly observed in the ear, eye, pharynx, larynx, and throughout the system, as manifested by constitutional depression, nervous erethism, mental disturbances, and derangement of the various viscera.

CROUPOUS RHINITIS.

By F. H. BOSWORTH, M.D.

A MORE correct understanding of inflammatory diseases involving the nasal passages will, I think, be arrived at if we adopt that classification in which the term used to designate a disease also indicates its pathological character. Thus, when we speak of simple rhinitis, we allude to a catarrhal rhinitis, or an inflammation characterized by a fluid discharge; when we speak of croupous rhinitis, we have to do with an inflammation characterized by a deposit, on the external surface of the mucous membrane, of a fibrinous exudation, or false membrane; when we speak of diphtheritic rhinitis, we allude to that form of inflammation characterized by the development of a false membrane which not only lies upon the surface, but also infiltrates the tissues of the mucous membrane down to its deeper layers. The term croupous rhinitis, then, we use to designate that form of acute inflammation of the mucous membrane lining the nose which is characterized by the formation on its surface of a fibrinous exudation.

The disease is met with both among children and in adult life. In children it runs a somewhat more protracted course and is attended with symptoms of a graver character, although the disease is rarely in itself a dangerous one. From an examination of the literature of the subject we would gain the impression that it is an exceedingly rare affection, for we find few writers alluding to it; and yet, I think, it is far more common than is usually supposed. Moreover, the literature of the subject is somewhat vague and indefinite, for we find Fränkel[1] referring to it as a complication of diphtheritic disease of the nose, thus confusing two entirely separate and distinct diseases, an error which Schüler[2] also falls into in reporting a case of the disease, still using the term diphtheritic. Cohen,[3] on the other hand, while recognizing it as a disease distinct from diphtheria, describes it as a complication of acute coryza. Later, however, we find Moldenhauer[4] recognizing it as a distinct disease, and giving us an excellent description of its course and symptoms; in the same manner examples of the disease were reported by Hartmann,[1] Seifert,[2] and Ryerson.[3]

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[1] Ziemssen's Cyclopædia, Am. ed., vol. iv. p. 136.

[2] Jahrbuch für Kinderheilkunde, N. F., Jahrg. iv., 1871, p. 331.

[3] Diseases of the Throat and Nose, 2d ed., p. 332.

[4] Monatschrift für Ohrenheilkunde, No. 9, 1887.

Causation.—When we come to consider the causes of the disease, we enter upon the discussion of pathological problems by no means yet fully determined. I think, however, that the weight of clinical evidence is largely in favor of the view that a croupous exudation is a local manifestation of a general disease. Thus, a diphtheritic exudation we regard as local evidence of the general disease which we term diphtheria; and so a croupous exudation is to be regarded as the local evidence of a general disease to which we have as yet given no definite name. That this is true, I think, is fully evidenced by the fact that the disease is invariably attended with very great systemic disturbance and general febrile movement, far more so than would be commonly expected as symptomatic of a purely local inflammatory action. The prominent general condition, probably, is an excess of fibrin in the blood, which we may call hyperinosis, and which so far dominates the local inflammatory action as to change a simple catarrhal inflammation into one attended with an escape of fibrin from the blood, which changes the simple mucous discharge into a fibrinous exudation, and, as the result, the formation of a false membrane. Now, furthermore, I believe that a croupous exudation is primarily due to the deposit of a germ upon the surface of the mucous membrane, which, making its way into the blood, gives rise to this condition of hyperinosis, and which at the same time, also, has a certain influence, perhaps, in exciting the croupous inflammation at its point of entrance. Of this we have abundant clinical evidence in the fact that the surface of the tonsil is the most frequent site for the formation of any false membrane, whether croupous or diphtheritic, and in the ragged surface of the faucial tonsil we have the physical conditions which afford the most favorable site for the lodgement of a germ, in that it becomes entangled, as it were, in the crypts or innumerable open-mouthed follicles which are found in this region. Furthermore, that the disease should be exceedingly rare, as involving the nasal cavity, is accounted for by the fact that here we have to do with an absolutely smooth surface, which is constantly bathed in the profuse flow of serum which constitutes the respiratory exosmosis, and, still further, any lodgement of a germ on this surface is prevented by the constant to-and-fro current of air, and also by the ceaseless activity of the vibratory movements of the cilia.

At a rather interesting discussion on this disease which occurred at the sixtieth meeting of the German Naturalists and Physicians, Bresgen made the observation that not infrequently it resulted from the use of the galvano-cautery in the nose. I suspect that he regarded the superficial slough which resulted from the cauterization as a croupous exudation, a morbid condition essentially different. Hering and Schmithinson, in the same discussion, alluded to the insufflation of impure water as a cause of the disease, an observation which it would be rather difficult to verify. A croupous exudation undoubtedly occurs after operations in the nose which involve a section of the membrane, a condition having thus been established which favors its development,—viz., a cut surface. The disease, however, in this case differs essentially from the idiopathic attack of croupous rhinitis, in that the membrane shows no disposition to extend, although the attack is attended with quite as well marked evidences of systemic disturbance as in sporadic cases. I think, then, that we may consider that the disease is a germ-disease, caused in exceedingly rare instances by the germ lodging upon and making its way into the interstices of an apparently sound mucous membrane, and in other cases invited by the open-mouthed follicles of the cut surfaces in connection with operations in this region.

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[1] Deutsch. Med. Wochenschr, 1887, No. 9, p. 641.

[2] Münchener Med. Wochenschr, 1887, No. 38.

[3] New York Med. Record, July 30, 1887.

Pathology.—The essential pathological lesions observed in the mucous membrane proper are those of an ordinary acute rhinitis, and consist, briefly, of a hyperæmia of the blood-vessels both of the turbinated tissues and of the mucosa proper, together with a general increase of the nutritive processes met with in that disease,—namely, increased exudation of serum, and cell-proliferation. On the surface of the membrane, and somewhat loosely adherent to it, is found a false membrane, presenting the following characteristics: there is a basement-substance of fibrillæ of fibrin, and considerable granular fibrin. Entangled in this basement-substance we find numerous white blood-cells and epithelial cells, undergoing granular degeneration and coagulation necrosis; some red blood-cells are also to be observed. The membrane thus presents the ordinary characteristic appearances of a croupous membrane. The exudation occurs on the faces of the lower and middle turbinated bones, and, in aggravated cases, on the septum. I have never noticed any tendency to extension to the accessory sinuses, although their orifices are closed by the swollen nasal membrane, and distressing symptoms referable to those cavities may occur thereby.

Symptomatology.—A croupous rhinitis follows the rule observable in all cases of diseases of the upper air-passages characterized by the superficial deposit of fibrinous exudation, in that its onset is attended with well-marked evidences of general disturbance. In most cases the invasion is attended with a chill, although in many cases there is merely a chilly sensation. This is followed by general febrile motion, the thermometer, as a rule, on the first day marking a temperature of 102° to 103° F. The higher temperatures are not usually observed in the nasal disorder. In connection with the fever there is usually pain in the back, headache, depression of spirits, and the chain of symptoms which are embraced under the expression of general malaise. The high temperature lasts from one to two days, when it subsides, and the further progress of the disease is attended with a temperature of 100° to 101° F., or, in certain cases, even as low as 99° F.

The subjective symptoms, attended with sneezing and watery discharge, indicate apparently a cold in the head. This, however, is soon followed by the development of the croupous membrane, whose progress is very rapid, so that at the end of twenty-four to thirty-six hours it extends throughout the nasal cavity, resulting in complete stenosis. At this stage of the disease the discharge assumes a more muco-purulent character, although a well-developed pus-discharge is rarely present. The disease involves both nasal cavities alike, and, nasal respiration being thus arrested, the facial expression of the child presents the characteristic appearance which we all recognize in this condition, in that the open mouth and the apparent broadening of the bridge of the nose give rise to the characteristic vacant expression.

Moldenhauer,[1] however, states that in three or four cases under his own observation but one side was affected. This does not harmonize with my own experience, although complete stenosis exists in either case, for a croupous exudation in one nostril will give rise to stenosis of the opposite side by a swelling of the turbinated tissues. In most instances the membrane extends simply through the nasal cavities; occasionally, however, we find it involving the upper pharynx, and even the faucial tonsil, although in this region it assumes the character of a follicular tonsillitis rather than that of a false membrane.

Diagnosis.—The disease is easy of recognition in those cases in which the false membrane extends fully down to the muco-cutaneous junction. In certain instances, however, the deposit is confined to the upper portions of the nasal cavity, in which case the orifice of the nose is occluded by the profuse discharges of mucus and muco-pus which result from the inflammatory process. In those cases it will be necessary carefully to wipe away the accumulation, for the thorough inspection of the part; for it is a matter of importance that the condition should be recognized, and it should always be suspected in cases of apparently an ordinary acute rhinitis attended with marked general disturbance and high febrile motion. The inspection of the parts is not aided in any way by the use of cocaine, since the turgescence of the membrane does not respond to its local action in reducing vascular congestion. Our dependence here, then, must be entirely on the nicety of manipulation in removing the secretions, and the careful inspection of the cavity with a good light, the reflected rays of the sun being always preferred as the source of illumination. When brought into view, the false membrane presents the characteristic appearance of an ordinary croupous exudation, in that it is a clean, clear, white membrane, presenting no evidences of necrosis or necrotic process, such as is characteristic of diphtheria, but every appearance of vitality. Furthermore, on delicately manipulating the probe, it will be found that the false membrane can be lifted from the surface of the mucous membrane beneath, which then will be found absolutely intact. In other words, the removal of the false membrane is attended with no rupture of blood-vessels, as is characteristic of the diphtheritic membrane. There is this difference, however, to be recognized in a fibrinous exudation in the nose,—that the fibrin in the membrane is not very abundant: hence the exudation is of a softer and more friable character, and, instead of being peeled off, as it were, in a continuous layer, it is more liable to be broken up into small granular masses in its removal.

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[1] Loc. cit.

Prognosis.—A croupous exudation, in itself, is never dangerous to life, the only gravity which attends the disease being on account of its locality. Thus, when the larynx or trachea is the site of the deposit, it destroys life by suffocation; when, on the other hand, other portions of the air-passage become the seat of the deposit, it is a self-limited disease, attended with a certain amount of discomfort and the possible danger of subsequent impairment of health; but otherwise it is rarely a dangerous disease. When a croupous deposit occurs on the face of the tonsil, we recognize a very serious danger of a secondary deposit in the larynx. There is no clinical evidence, however, to show that a croupous rhinitis in any way tends to a secondary formation in the parts below. The disease runs a somewhat protracted course in children of from three to five weeks. Hartmann[1] states that its course is from five to eight days. I have not infrequently seen this short course of the disease in adults, and am disposed to think that Hartmann's observations were made on adult patients. I have never known so short a term, however, in young children, and doubt if it ever runs a shorter course than three weeks, and probably in many instances longer. Bresgen[2] also assigns a course of several weeks to the disease.

Treatment.—*Local Treatment.*—The tendency after removal of the membrane is to a redevelopment: hence the essential feature of local treatment should consist in some measure by which the membrane is not only removed but its further development arrested. For this purpose, probably we have no single drug which possesses the promptness and efficacy of the preparations of iron, and of these either the tincture or officinal persulphate may be used in full strength, provided the application is made with that nicety and delicacy of manipulation by which the unpleasant action of these drugs on the healthy structures may be avoided. The membrane, as before shown, is exceedingly soft and friable: hence it can be easily removed, with delicate manipulation, by a small cotton pledget on the end of the probe, care being taken to do no injury to the membrane beneath; the point being, that if blood-vessels are ruptured or injury done to healthy tissues, a certain danger arises of absorption of morbid material, which is always to be carefully avoided. As before stated, cocaine is not of much value in relieving turgescence, but certainly it should in all cases be made use of to accomplish such vascular contraction as may be possible, and, furthermore, to facilitate the further procedure by local anæsthesia. After the membrane has been removed, the inflamed surface beneath should be carefully brushed over with small pledgets of cotton soaked in either persulphate or tincture of iron. This manipulation is to be repeated daily, or, better still, twice daily, until the morbid process is brought fairly under control. Where the exudation presents as a thin, continuous membrane, it is oftentimes better to leave it *in situ* without removal, and simply destroy its activity by saturating it with one of the preparations of iron in the manner before stated, thus substituting for an actively diseased condition an inert film, for, as we know, the iron absolutely destroys all activity in fibrinous deposits. This film, lying upon the mucous membrane, serves to protect it somewhat and probably to prevent, to an extent, a recurrence.

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[1] Loc. cit.

[2] Loc. cit.

General Treatment.—As before suggested, the essential systemic condition in these cases is one of hyperinosis, and possibly we possess no remedy more active in controlling this condition than the tincture of iron. Hence in all cases of croupous rhinitis this should be administered for its systemic action, as follows:

℞ Tinct. ferri chlor., ʒ ii;
Glycerini, ad ℥ ii.
M.
Sig. A half-teaspoonful every four hours.

In addition to this, and especially in young children, I think there can be no question that mercurials possess a certain power in controlling a fibrinous exudation. Hence they should be administered in pretty full doses in connection with the iron, until their action has been thoroughly tested. For this purpose, probably we possess no remedy better than the mild chloride:

℞ Hydrarg. chloridi mitis, gr. xx;
Sacch. lact., ad ʒ ii.
M. et div. in chart. no. xx.
Sig. One to be given every four hours.

Aside from these measures, the further management of the case will be based on those general rules which govern the control of febrile movement, together with building up the system, where required, by administration of tonics, careful attention to the diet, and relief of such torpid condition of the bowels as may exist. It should be borne in mind, of course, that in all cases where iron is administered it will ordinarily be necessary to administer laxatives, and for this purpose preference should be given either to castor oil or to one of the preparations of rhubarb.

RHINITIS ATROPHICA.

By WILLIAM CHAPMAN JARVIS, M.D.

Synonymes.—Atrophic or Dry nasal catarrh, Rhinitis atrophica simplex, Rhinitis fœtida atrophicans, Ozæna.

Definition.—A chronic affection of the nose, characterized by the shrinkage or atrophy of the pituitary membrane without ulceration, and accompanied with the formation of mucus or muco-purulent crusts, which, as a rule, though not invariably, give rise to an offensive odor.

Etiology.—While a cause of rhinitis atrophica is to be found in constitutional syphilis and scrofulosis, instances of this kind are comparatively few in number, and the careless practice of attributing the affection to these dyscrasiæ is becoming more infrequent with the increase of our knowledge of the local manifestations of the disease. The evidence that long-existing moist catarrhs commencing in early life may develop the dry form of the disease, is overwhelming. Chiari, in one hundred and thirty-seven cases of atrophic rhinitis, found that in one hundred and three the disease began before seventeen, and in most of these in the fifth and sixth years of life. Mackenzie noticed that the moist rapidly passed into the dry form at puberty. Schäffer diagnosticated hypertrophic rhinitis in a boy aged five, who presented himself five years later with a typical rhinitis atrophica. Failure to properly treat the humid form of the disease at the most favorable period in early life, also explains the frequency of the affection, for, as Ziemssen remarks, "many physicians regard a subacute or chronic nasal catarrh of a child as an ailment that is neither worthy of nor amenable to treatment."

Zaufal asserts that rhinitis atrophica owes its existence to an inordinate breadth of the nostrils in the new-born and atrophy of the inferior turbinated bone. Although this is an extreme view, it cannot be doubted that many cases of atrophic rhinitis owe their origin to an inherited vicious formation of the nasal chambers, more readily discoverable at or just before the period of puberty. As several members of the same family may be affected, the manifestations of this condition may be mistaken for those of an inherited constitutional disease. Löwenberg claims to have discovered a coccus in atrophic rhinitis which cannot be classed as putrefactive in character, and is found only upon the diseased mucous membrane.

Pathology.—Rhinoscopic inspection reveals the presence of incrustations closely adherent to the inner walls of the nose, which, upon being removed, expose either an inflamed, irritated mucous membrane, or one exhibiting a pale, smooth surface. The meatuses are either obliterated or appear unusually shallow, and the turbinated ridges are greatly reduced in size or their outlines may be with difficulty discerned. The disease may be largely confined to the naris proper or may involve the accessory cavities of the nose. Ozæna is evidently principally caused by the decomposition of muco-purulent or fatty matters in the presence of a small amount of moisture, or by a fetid exhalation (Moure). There is a marked diminution in the quantity of the secretions, which are, furthermore, more purulent than mucous in character.

Microscopical examination has demonstrated cornification of the epithelium and the formation of fat-globules (Krause), cirrhosis of the submucous cellular tissue (J. Mackenzie), atrophy of the glandular follicles (Gottstein), and disappearance of the venous sinuses of the turbinated tissues (Bosworth).

Diagnosis.—While it is very easy to diagnosticate the existence of an atrophic rhinitis in adults, on account of the amplitude and distinct outlines of the nasal chambers, some difficulty may be experienced in differentiating the atrophic from the hypertrophic form of the disease in early childhood. The affection will be found to be, as a rule, clearly defined, midway between the interval of five and fifteen years. Inasmuch as the manifestations of rhinitis atrophica possess a marked individuality, the exercise of care will usually enable one readily to differentiate the affection from all other forms of nasal disease, and there seldom exists, within the period given, any good reason for confounding this malady with hypertrophic rhinitis.

The distinguishing structural features of atrophic rhinitis are smoothness of the pituitary membrane, loss or reduction of the outlines of the turbinated bodies, abnormal spaciousness of the nasal chambers, shrinkage of the adenoid tissues in the vault of the pharynx, and pharyngitis sicca. The secretory peculiarities are the formation of crusts and nasal moulds, pronounced fetor of the nasal discharges (ozæna), and marked diminution and thickening or concentration of the secretions. The prominent symptoms are sensation of dryness, nasal obstruction from the accumulation of scabs, headache, a stench compared by the French to that of crushed bed-bugs (*punaisie*), excoriations, and hemorrhagic abrasions caused by scabs.

Prognosis.—While childhood is a period most favorable for the successful treatment of hypertrophic rhinitis, it offers the only opportunity to accomplish a cure in the atrophic form of the disease. Even in cases where the mucous membrane has lost a portion of its secretory power, persistent treatment, favored by the developmental processes, may result in a restoration of this function. When the disease is of several years' standing or the atrophic condition is extensive, while it is safe to promise permanent relief from the fetor, scabs, and dryness, provided treatment is persisted in, it will be impossible to extend more than the hope of a probable cure in certain more favorable cases. The transitional period between the moist and dry forms of the disease naturally offers the best results in the radical treatment of the affection. The fact that this change can occur, and the difficulty experienced in determining just when it takes place, should urge the adoption of prompt measures in the treatment of all forms of moist catarrh.

Treatment.—Recognizing and studying atrophic rhinitis as existing in two distinct forms,—namely, with and without the symptom ozæna,—the treatment must be regulated in accordance with this division. As already explained in the paragraph devoted to the pathology of this disease, atrophic rhinitis, unaccompanied by an ozæna or stench, is seldom found in childhood,—so rarely, in fact, that the treatment of rhinitis atrophica simplex may be safely disposed of with this explanation.

In the first place, it may be well to outline briefly the special indications for local and constitutional treatment, as determined from a clinical and pathological stand-point.

The measures adopted for the local treatment of the affection have for their object, first, the loosening and removal of the intra-nasal incrustations and thickened secretions; second, the prevention or retardation of the return of these conditions, and the maintenance of the nasal chambers in a state of asepsis; and, third, the improvement of the general health.

The first of these indications is accomplished by the judicious employment of detergent, antiseptic douches, reinforced by the loosening action of the brush or cotton probe. The choice of lotions for this purpose is, with perhaps an occasional modification in regard to strength or increased antisepsis, almost identical with that given for the treatment of rhinitis hypertrophica. The slight alteration is only required to render the antiseptic action of the fluids more searching in their elimination of an additional feature of this affection, namely, the fetor. They may be added to the cleansing solutions referred to in hypertrophic rhinitis in the following proportions of increased strength to each ounce of the medicated fluids: carbolic acid, gr. ¼–iv; salicylic acid, gr. i–iv; salicylate of sodium, gr. v–x; sulpho-carbolate of zinc, gr. ss–ii; solution of chlorinate of sodium, ʒss–ʒi; benzoic acid, gr. ss; benzoate of sodium, gr. i–x; thymol, gr. ¼–i; permanganate of potassium, gr. i–v; and bichloride of mercury (1 to 10,000). The proportion of each of these agents will, of course, vary with the condition of the patients, which may when the ozæna is slight require only a small percentage, or when the sensitiveness of the mucous membrane approaches the normal state, which is rarely the case, weak solutions are demanded. In marked cases of atrophic rhinitis, sensation is often so greatly impaired that what would in the normal or hypertrophied nostril constitute a most painful and long-continued irritant amounts to a mild degree of stimulation. Glycerin, when employed in conjunction with these antiseptic nasal washes in the proportion of from fifteen minims to a drachm to the ounce, will be found to be a most valuable agent in promoting the removal of crusts, by its softening and solvent action, and to soothe the irritated and ofttimes inflamed mucous membrane. Furthermore, these washes should invariably be employed at an elevated but comfortable temperature, since the warmth facilitates the disintegrative action of the solutions.

Of great importance is the means employed to render the application of these antiseptic detergent fluids effective. The quantity of the liquid must be copious, and, different from the hypertrophic form of the disease, considerable force is required to project it effectively through the nostrils. For this reason, the hard-rubber post-nasal syringe is to be preferred above all the forms of nasal douches; nor need one fear evil consequences from entrance of the fluid into the middle ear in typical cases of rhinitis atrophica, since the abnormally spacious nasal chambers permit the easy exit of the injected fluids. The manner in which the syringe is manipulated has been already explained in the paragraph on the treatment of hypertrophic rhinitis. Where the nostril is partly obstructed by reason of deflection of the septum, the point of the syringe should be carried into the choana, past the Eustachian orifice, of the narrow nostril, and it will sometimes be found advisable to correct these septal deviations to facilitate flushing of the contracted nostril.

Sometimes the tenacious crusts adhere with a firmness that resists the action of detergent fluids and necessitates the employment of the probe or forceps to loosen them from their attachment. This condition, however, as a rule, presents itself only at the first visit, and when observed subsequently is the result of neglect, since it cannot recur if the proper precautions have been carefully carried out. One of these precautions consists in the daily use of the syringe, at the hands of the physician, parents, or exceptionally the child. Parents, as a rule, readily learn to use the post-nasal syringe, and they should be instructed to wash out the nasal chambers at least twice daily,—namely, in the morning and evening. Should they fail to acquire the requisite dexterity or be prevented from accomplishing this result by the rebelliousness of the child, anterior nasal douching may be resorted to as a less effective but a necessary substitute. For this purpose a hard-rubber ear-douche may be employed, or the nozzle of an air-bag may be fitted to the rubber bulb of a Warner syringe. Warner's post-nasal douche may also be conveniently employed by the child's parent as a substitute for the hard-rubber syringe.

The second proposition, that of retarding and preventing the formation of intra-nasal incrustations or diminishing the accumulation of the fetid secretions, is a very important one, and can be satisfactorily answered. Nothing so effectually prevents this tendency of the secretions to inspissate, and in this manner generate crusts, as the application of suitable unguents. Many varieties of these agents have been recommended for this purpose, but the mention of a few of the most common—namely, vaseline, lanoline, lard, cacao butter, and gelato-glycerin—will suffice to give an idea of their character. Vaseline forms a useful example of this class of medicaments. After the nostrils have been thoroughly cleansed in the manner already explained, this lubricant may be carried into the nostrils upon a cotton probe, brush, or feather, or in a much more thorough and agreeable manner by utilizing the nasal spray. When the latter method is employed, the yellow vaseline or more elegant white preparation is gently heated and poured into the spraying reservoir, which may be the bottle of an ordinary hand-atomizer. When applications have to be repeated, it is only necessary to allow the spray reservoir to remain for a few minutes in a vessel of hot water, to reduce it to a state of fluidity. Vaseline when freely sprayed into the nostrils is naturally more searching in its reach than any of the hand-applications employed for this purpose. It rapidly congeals after touching the pituitary membrane, thereby coating the nares with a delicate unctuous film. This film effectually prevents the collection, drying, and adherence of the secretory ooze upon the exposed surfaces, and proves very grateful to the irritated and inflamed mucous membrane. With slight differences in the method of application and in action, the remaining agents are indicated for the same conditions mentioned for the employment of vaseline.

Another class of remedies which have proved very serviceable for preventing the formation of incrustations in the markedly atrophic dry nose of adults, and which may prove similarly serviceable in the more advanced forms of rhinitis atrophica in children, is the employment of local stimulation. This measure is more particularly indicated in that form of the disease which expends itself principally upon the lining membrane of the nostrils and but slightly upon the accessory nasal sinuses. Serpentaria, galanga (Bosworth), red gum (Mackenzie), white hellebore, nitrate of silver (Michel), eucalyptus (fluid), and tampons (Gottstein and Woakes) are examples of these agents. They act by stimulating the dormant secretory follicles or by increasing the energy of those which have survived the destructive advance of the atrophic processes, their efficiency being due to the flushing of the nasal chambers brought about by the artificial flux. As a natural consequence of this moistening of the mucous membrane, scales cannot form, and the nasal detritus finds a more ready exit from the nostrils, thus, as is especially the case with Gottstein's plugs, preventing the development of a stench from the decomposition of incarcerated nasal secretions. The powders, reduced to a state of exceedingly fine subdivision, are best applied by means of Ely's powder-blower, and only after careful cleansing of the nasal chambers has been effected. The stimulating action of eucalyptol is obtained by spraying the oil into the nostrils. Gottstein's tampons and Woakes's plugs owe their efficiency to the generation of a nasal flux by the prolonged presence of packed cotton-wool (G.) and medicated wool (W.) in contact with the adjoining surfaces.

Finally, constitutional measures may be required to meet the dyscrasic features sometimes present in the fetid forms of atrophic rhinitis,—namely, non-ulcerative syphilis and scrofula,—and to improve the general health of the patient by the improvement of his manner of living and environment. The first object is accomplished by the administration of the proper antisyphilitic specifics, cod-liver oil, the hypophosphites, iron, syrup of the iodide of iron, quinine, etc.; the second, by the employment of proper hygienic precautions, attention to diet, change of climate, and improved sanitation.

Ulcerous coryza (Robinson) or rhinitis complicated by the presence of ulcerations, syphilitic and otherwise, may be either atrophic or hypertrophic in character, and, inasmuch as the therapeutic measures adopted for its cure have for their object more the healing of the ulcers than the treatment of the coexisting or consequent catarrh, their consideration would not be strictly proper under the head of rhinitis atrophicans.

PURULENT RHINITIS OF CHILDREN.

By F. H. BOSWORTH, M.D.

THIS term is used to designate a form of catarrhal disease which is met with exclusively in young children and is characterized mainly by a more or less profuse secretion of muco-pus from the nasal passages. The disease, I think, never has been definitely described in current literature, but in my own experience it has been met with so very frequently as to warrant its description as constituting a definite form of inflammatory action involving the nasal mucous membrane. An ordinary acute or chronic catarrhal inflammation of the mucous membrane is characterized by an apparently excessive discharge of mucus, together with a certain amount of turgescence of the mucous membrane and impairment of its function, the discharge being usually an excess of the ordinary secretions of the membrane, surcharged somewhat with desquamated epithelial cells so as to render it slightly opaque in color. In the disease under consideration, we have a chronic inflammatory process in which, while there is a certain amount of turgescence of the membrane, together with increased secretion of mucus, the prominent feature of the disease consists in a certain activity of cell-proliferation, involving largely the epithelial layer of the membrane, whereby the mucus becomes greatly surcharged with epithelial cells, which, owing to the rapidity of their desquamation, fail to attain full maturity, or, in other words, become merely pus-corpuscles, which thus, being generated in large numbers, permeate the mucous secretion and convert it really into a yellow, somewhat thick, purulent discharge.

In examining the literature of the subject, we find the term purulent rhinitis occasionally making its appearance, though in a somewhat vague and indefinite manner. Mackenzie[1] confines the use of the term to the acute form met with in infancy, and usually attributed to infection from the genital passages of the mother, although he questions the accuracy of this view; while under the chronic form he[2] would seem to refer to that curious affection, first described by Stoerck as occurring as a local disease among the Poles, which consists in the development of a purulent discharge, mainly as the result of uncleanly habits,—a disease characterized by no injection of the membrane, but one which runs an essentially chronic course, and is said to extend to the lower air-passages, giving rise to dyspnœa, in one case tracheotomy having been required. Fraenkel[1] confines the use of the term to the acute variety of the disease referred to by Mackenzie; while Cohen,[2] in his chapter on chronic nasal catarrh, alludes rather casually to a purulent form of the disease which occurs in infancy and runs a somewhat prolonged course, resulting in ulceration and necrosis,—probably referring to syphilitic disease. Beverley Robinson, Sajous, Brown, and others make no reference whatever to the purulent form of nasal disease. It is a very notable clinical fact that inflammatory processes, not only of the mucous membrane, but also of other tissues of the body, in children show a tendency to involve the epithelial structures, while in adult life this tendency seems to disappear, and the connective-tissue structures are peculiarly liable to become involved in inflammatory action. This is strikingly evidenced in diseases of the upper air-passages: thus, in child-life a catarrhal inflammation of the nasal mucous membrane proper is somewhat rare, but it is the lymphatic structures that are especially liable to diseased action, such as the pharyngeal and faucial tonsils. This clinical fact I do not find recognized in literature, and yet Wagner[3] would seem to suggest it, for he makes the statement that "during childhood the skin and mucous membrane are excitable; the function of the lymphatics is more prominent; the quantity of lymph is increased; the lymphatic glands at this time have the greatest development." We find, then, that an inflammatory process involving the mucous membrane proper does not, as an ordinary acute rhinitis, manifest itself prominently in the turgescence of the blood-vessels with the secretion of mucus, but this tendency in child-life to the involvement of epithelial structures dominates the process so far as to cause a form of inflammation in which the superficial layer of the mucous membrane becomes actively involved in the inflammatory action, and hence there arises a morbid process in which epithelial desquamation becomes the prominent feature. For the better understanding of the subject under consideration, I think it should be made clear at this point just what its clinical significance is, and what ultimate results are to be expected if the disease remains unchecked.

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[1] Diseases of the Throat and Nose, vol. ii. p. 294.

[2] Loc. cit., p. 335.

It is essentially a chronic disease, and runs an exceedingly protracted course, extending over from five to fifteen years, in all cases probably commencing in childhood. Its essential feature, then, consists of a rapid cell-proliferation, resulting in profuse cell-desquamation. We have here commencing a process which, at its onset involving only the superficial layer of epithelium, gradually extends to the gland-structures of the membrane. In the early stages of the disease the desquamation of epithelium is fully compensated for by cell-generation: hence the membrane proper suffers no loss. As time elapses, however, there comes a period when the desquamation of epithelium exceeds the cell-production, and the membrane suffers. This process is not attended with any deleterious results as long as the process confines itself to the superficial layer, but sooner or later it extends to the epithelium lining the glands and follicles of the membrane, a process which, as it goes on, finally leads to a condition in which certain of the glands and follicles of the tissue become denuded of their epithelium. The consequence of this is that the gland-structures collapse. The result which follows this loss of the secreting apparatus of the membrane is very clear: a less amount of healthy mucus is secreted. Hence that which is poured out on the surface of the mucous membrane, filled as it is with desquamated epithelial cells, becomes less fluid, shows greater tendency to inspissation, and finally, when the loss of gland-structure has gone on to a serious extent, there comes a time when the secretion becomes exceedingly thick and contains a comparatively small proportion of water, and hence dries rapidly, forming crusts upon the surface of the membrane. We now see clearly what the clinical significance of a purulent rhinitis is. It is the early stage of atrophic rhinitis or ozæna. It is usually stated that atrophic rhinitis is a late stage of hypertrophic rhinitis. Probably in all medical literature there is no single statement so utterly unwarranted and based on such entirely incorrect clinical observation. The hypertrophic form of rhinitis is a hypertrophic process from its onset. Atrophic rhinitis follows upon purulent rhinitis in the manner above stated, as soon as the secretion in the purulent affection becomes so thick that it dries readily on the convex surfaces of the turbinated bones. We thus have crusts adhering closely upon the turbinated bones, and contracting slightly, as the result of which they lodge in the cavity of the nose and remain for from twenty-four to forty-eight hours or even longer. Now, composed largely as they are of animal matter, the natural result is a certain amount of decomposition, and the offensive odor of ozæna is developed. The further progress of the disease consists of a true atrophic process of the membrane, involving finally the bone also. There are two features which lead to this condition. The collapse of the glandular structures, which is attended with a form of cirrhosis, as it were, in the mucosa proper, by reason of which the circulation of blood is notably interfered with, is perhaps the most active factor in producing this condition; but, in addition to this, the crusts which form on the surface of the membrane, contracting slightly, add to this interference with the circulation in no slight degree. Now, as the deep layer of the mucous membrane is the periosteum, anything which interferes with the flow of blood through this deep layer necessarily interferes with bone-nutrition. Hence the latter stage of this disease, which commencing as a purulent rhinitis develops subsequently into ozæna and still later into an atrophic rhinitis, consists simply in an atrophy of the turbinated bones due to a shutting off of their blood-supply, this latter condition being the last stage of the disease which we call ozæna, or more properly atrophic rhinitis.

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[1] Ziemssen's Cyclopædia, vol. iv. p. 139.

[2] Diseases of the Throat and Nose, 2d ed., New York, 1875.

[3] Manual of General Pathology, New York, 1876.

Causation.—The disease is essentially a local one, and is in no way connected with any peculiar diathetic condition, nor is it the result of impairment of the general health. Writers on ozæna frequently describe it as due to a scrofulous or tubercular diathesis. My own experience teaches me that the sufferers from this disease, whether in its onset as a purulent rhinitis or in its later stages, enjoy vigorous health. We simply say, then, as regards causation, that it is probably due to some errors in hygienic surroundings, insufficient clothing, or improper diet, which lead in child-life to a habit of taking cold, which at this time of life, as we have seen above, tends to manifest itself in the peculiar form of inflammation here described. Undoubtedly in many cases it has its origin in an attack of measles, scarlet fever, or some of the other exanthemata, whose development and course are so frequently attended with catarrhal inflammation of some portion of the upper air-passages.

Symptomatology.—The prominent symptom of the disease consists of a discharge from both nostrils, of a somewhat clear, yellowish, thick, muco-purulent catarrhal secretion which shows a disposition to form crusts in the lower portion of the anterior nares, or unsightly accretions around the margin of the nostrils at the muco-cutaneous junction. If the child is old enough to use the handkerchief, the discharge is expelled in this way in considerable quantities, staining the linen a bright yellow. If, on the other hand, it remains in the nasal passages, it accumulates in such a way as to give rise to notable stenosis. In addition to this, as in all chronic inflammatory affections, there is a special liability to take cold, and the child suffers in this manner from even slight exposures. During an acute exacerbation the amount of discharge is increased, while at the same time the mucous membrane is notably swollen and the nasal stenosis markedly increased. In fact, the child suffers from an ordinary acute coryza. At other times, however, there is no notable stenosis, the morbid process confining itself, as we have seen, entirely to the superficial layer of the mucous membrane, while the large venous plexuses beneath are not notably involved. Of course the amount of blood sent to the part is larger than normal, and yet there is not that active vascular plethora that characterizes a simple chronic rhinitis. The morbid process involves the middle as well as the lower turbinated bodies: hence we might expect that a certain amount of hypersensitiveness would be present, as indicated by sneezing, etc. I am disposed to think, however, that sensibility of the membrane is but slightly diminished, if at all changed. Hence the subjective symptoms other than the nasal stenosis are not prominent. As the result of stenosis, and its consequent mouth-breathing, pharyngeal and laryngeal irritation may result in a cough. This usually is of a dry, hacking character, and ordinarily not attended with any expectoration. Croupy attacks, or any other evidences of laryngeal irritation, would only be the result of nasal stenosis.

Diagnosis.—A diagnosis in these cases is of the greatest importance, in view of the fact that if the disease runs on to the stage of crust-formation or ozæna we have to do with an affection usually not amenable to treatment. Fortunately, its recognition is comparatively easy, as there are few diseases with which it may be confounded. Syphilitic or scrofulous disease of the nose is attended with pus-discharge, the result of ulceration and necrosis. In these cases the discharge, therefore, would be mingled with masses of black necrotic tissue or portions of bone, which would be at the same time attended with an intolerably offensive odor which could never by any possibility be mistaken for the odor of simple purulent rhinitis. In addition to this, there would be the other evidences of poison in the system, such as a general cachexia, skin eruptions, or other concomitant symptoms. Moreover, syphilitic disease of the nose is usually unilateral, while the affection under consideration is always bilateral.

Young children are exceedingly prone to insert small bodies into the nostril, but a child usually contents itself with inflicting this injury upon one nostril. The presence of this body gives rise to a more or less profuse pus-discharge, occasionally mingled with blood from the side affected. The diagnosis, however, in this case should always be simple, as purulent rhinitis is invariably bilateral. An examination by gross inspection and the probe should also be sufficient to make the diagnosis clear. Disease of the accessory sinuses occasionally occurs in quite young children. Here, also, we have a discharge of pus identical in all its features with those of purulent rhinitis, but the fact of its occurring in one side would eliminate any error in diagnosis.

If we make an examination anteriorly, we find the mucous membrane somewhat congested and of a dark-reddish color. Never, however, do we notice the active plethora of acute rhinitis. Furthermore, the membrane is covered with flakes and masses of yellowish mucus coating the lower turbinated bones and lying in masses on the floor of the nares. An examination of the pharynx, also, will usually show that a certain amount of secretion has made its way to this region and lodges upon the posterior wall of the pharynx in large shreds, which coat its walls and hang down between the pharynx and soft palate. The source of this, of course, might be in an enlargement of the pharyngeal tonsil. The peculiar character of the voice and peculiar facial expression are ordinarily sufficient for a recognition of this disease. If there be any doubt, an inspection with the rhinoscopic mirror or by a digital examination will reveal the source of the secretion. Blennorrhœa usually occurs at childbirth, and is characterized by a profuseness of discharge and activity of inflammatory process such as to render its recognition comparatively easy. Furthermore, as we know, a blennorrhœa of the nose in childhood rarely exists without the eyes sooner or later becoming affected.

Course.—The disease commences at from three to six years of age, and runs a course of about ten or eleven years before the crust-formation sets in.

In an examination of eighty-three cases of atrophic rhinitis of which I have records, in fifty-one the disease commenced as a purulent rhinitis between the fifth and sixth year of age, in two cases it commenced in the second year of life, while in four it commenced after ten. In all cases where the records are complete, it was clearly made out that the early stage of the disease was a purulent discharge.

Prognosis.—I regard the disease as a curable one if recognized before crust-formation has set in, for, if my views as regards its pathology are correct, we must recognize the fact that, while the discharge remains fluid, or, in other words, before crust-formation has set in, the glandular structures are not seriously involved, and that the disease is still confined to the superficial layers of the membrane and entirely within reach of remedial measures.

Treatment.—This is one of the diseases which is thoroughly amenable to local treatment, and that of an exceedingly simple character, the essential feature being that the cavity shall be thoroughly cleansed and subsequently subjected to the local action of some simple astringent. For cleansing purposes we may use one of the following:

℞ Acid. carbol., gr. iii;
Sodii bicarb., gr. xii;
Sodii biborat., ʒss;
Glycerini, ʒvi;
Aquæ, ad ℥vi.

℞ Listerini, ℥ss;
Sodii biborat., ʒss;
Glycerini, ʒvi;
Aquæ, ad ℥vi.

℞ Thymol., ♏xx;
Sodii chloridi, ʒss;
Sodii benzoat., gr. xx;
Aquæ, ad ℥vi.

℞ Ichthyol., gr. i;
Potassii chloridi, ʒss;
Liquor. calcis, ad ℥vi.

This should be applied twice or, if necessary, three times daily, at the hands of the nurse or attendant, by means of some simple hand atomizer, the spray being thrown repeatedly into one and then into the other nostril, the child being directed to blow the nose thoroughly after its application, until the parts are thoroughly cleansed. Fortunately, the disease occurs only in children who are competent to carry out this procedure. In very young children who have not learned to blow the nose considerable difficulty would be experienced, and it might be necessary to make use of the nasal douche, which requires no effort on the part of the patient, or, possibly, to employ a simple ear-syringe. After the parts have been thoroughly cleansed, an astringent should be used, as follows:

℞ Zinci sulphocarb., gr. xx;
Hydrarg. chlor. corros., gr. $\frac{1}{5}$;
Aquæ, ad ℥iv.

℞ Acidi borac., ʒii;
Aquæ, ad ℥iv.

℞ Acidi salicylici, gr. vi;
Aquæ, ad ℥iv.

To either of the above may be added with benefit any of the simple astringents, or these may be used alone, in order of preference as follows: glycerole of tannin, one drachm to the ounce; argenti nitratis, three grains to the ounce; zinci sulphatis, three grains to the ounce; cupri sulphatis, two grains to the ounce; aluminium aceto-tartrate, ten grains to the ounce.

As before stated, the disease is purely a local one, and the patients usually enjoy perfect health. Hence there is no special indication for the use of internal medication. It is of the greatest importance, however, that certain general hygienic rules should be observed in the management of these cases, such as the daily administration of a cold sponge-bath to the waist, together with careful attention to the diet, to the sleeping-apartments, and especially to the clothing. In all cases I think the underwear should be of pure wool, and worn summer and winter, as we recognize a notable liability in these cases to taking cold, and perhaps for the control of this disease we know of no measures comparable to the use of all-wool underwear in connection with the daily administration of the cold sponge-bath.

DISEASES AND INJURIES OF THE PHARYNX.

BY E. FLETCHER INGALS, M.D.

Acute Sore Throat.—*Synonymes.*—Pharyngitis, Cynanche pharyngea, Angina erythematosa, Angina catarrhalis, Angina simplex, etc.

This is a simple inflammation of the mucous membrane of the pharynx, palate, and tonsils, usually terminating in resolution, but in some individuals leaving a predisposition to future attacks which finally terminate in chronic inflammation. It occurs most frequently in children or in young adults, but may be met with at all ages. Among adults it is more frequent in those who follow sedentary occupations, and in subjects of syphilis or those who have been mercurialized. The throat is found congested in various degrees, sometimes in limited patches, at other times diffused over the whole surface. The mucous membrane is swollen, and in severe cases the uvula is œdematous.

Etiology.—It is caused by exposure and changes in temperature, especially among those who are poorly fed and clothed and who live in badly-ventilated apartments. It seems in some instances to be due to a scrofulous or rheumatic diathesis. It is sometimes epidemic.

Pathology and Pathological Anatomy.—The blood-vessels are dilated, and there is more or less inflammatory deposit in the submucous tissues, but the glandular structures seem to be most involved.

Symptomatology.—The affection is usually ushered in with slight fever attended by headache and heat of the skin, pain and itching of the throat radiating to the ears, sometimes a feeling as of a foreign body in the throat, and a frequent tendency to hawk and expectorate. In the more severe cases there is frequently at first a pronounced chill followed by fever, with a temperature ranging as high as 103° F. The constitutional disturbance is much more marked in children than in adults. The hearing is frequently slightly impaired, and the voice often has a nasal intonation. There is often a sensation of dryness and stiffness in the throat, which upon deglutition may become actual pain, especially when the inflammation is most marked in the upper part. When the inflammation extends to the lower part of the pharynx and to the larynx, the voice becomes husky, the tendency to hawk and hem is increased, and there is slight expectoration of tenacious mucus, which later becomes muco-purulent in character. The breath is foul, the tongue coated, the appetite poor, the bowels usually constipated, and the urine high-colored and loaded with urates.

Upon inspection of the throat, the congestion is found to involve the pharynx and usually the posterior pillars of the fauces and the soft palate; sometimes the anterior pillars and the tonsils are also involved. The veins are frequently seen to be enlarged, and the cervical glands are often swollen and painful.

Diagnosis—The only affection with which this is likely to be confounded is simple tonsillitis. The diagnosis is readily made after a few hours, from the fact that in tonsillitis the swelling of the glands is much more pronounced.

Prognosis.—The affection usually terminates in resolution in six or seven days, though a few fatal cases have been reported, principally from extension to the larynx. In some instances the patients are rendered peculiarly liable to renewed attacks.

Treatment.—Persons subject to this affection should be kept, so far as possible, in an equable temperature, excessive cold and overheated rooms being avoided. Cold sponge-baths daily render them less susceptible. At the beginning of the attack hot foot-baths are recommended. The inflammation may frequently be subdued by constant sucking of ice, in other cases by the frequent use of hot inhalations, and by either cold or hot compresses applied externally. Small doses of opiates may be given to relieve pain, and, where there is much fever, antipyrin, antifebrin, or aconite may be administered in appropriate doses. A spray of cocaine has also been recommended to relieve the pain, but its effects are so transitory and the dangers of an overdose in children are so great that its use is not advisable.

Where a rheumatic diathesis is present, guaiacum in the form of lozenges is found beneficial, but usually children object to the taste; therefore salicylate of sodium in syrup of lemon is more satisfactory. The bowels should be kept open with saline laxatives. Quinine, arsenic, or nux vomica, or these combined, are usually indicated. In excessive œdema of the uvula, scarification is indicated; but removal of the relaxed tissue should not be practised until the acute stage is passed, because of the tendency to sloughing. Astringent and caustic applications and strong counter-irritants are usually harmful. Fluid or semi-solid food should be given at regular intervals.

Erysipelatous Sore Throat.—This is a rare affection of the mucous membrane of the throat, generally associated with facial erysipelas. It is characterized by inflammation of the mucous membrane and subjacent tissues.

Pathology and Pathological Anatomy.—Cornil makes three divisions of this affection: first, erysipelas with simple redness, in which there is a diffused inflammation and the tissues are of a deep livid red and shining appearance, with more or less swelling; second, erysipelas with phlyctenulæ, in which vesicles appear, varying from the size of a pin's head to half an inch in diameter,—having the appearance of herpes and filled with serum or pus,—which rupturing leave yellowish-white patches of soft tissue that are easily torn from the tissue beneath; third, erysipelas terminating in gangrene, in which there is a dark pultaceous appearance with a gangrenous odor.

Etiology.—This affection is due to the same cause as cutaneous erysipelas. It is frequently endemic or epidemic.

Symptomatology.—This disease usually follows external erysipelas, and is ushered in by stiffness of the jaw and dryness or stinging pain in the throat, which is increased by deglutition. It is usually attended by more or less difficulty in breathing. Pain in the stomach and nausea are frequent symptoms. The temperature sometimes rises as high as 104° F., even before the efflorescence appears in the throat, and it may continue thus for three or four days. The submaxillary and cervical glands are frequently swollen. The difficulty in swallowing is due partially to the pain and partially to paralysis of the muscles. When the palatine muscles are affected, regurgitation takes place through the nose; if the pharyngeal muscles alone are affected, it takes place through the mouth on attempted deglutition. Upon inspection of the throat, the tissues are found swollen, of a dusky-red hue, or dotted with vesicles filled with serum, pus, or blood. In the gangrenous cases there is a dark pultaceous appearance and the characteristic odor is present.

Diagnosis.—In the absence of external erysipelas the diagnosis would be difficult.

Prognosis.—This is a grave disease, about one-half of the cases proving fatal. Death not infrequently occurs within two or three days. In those cases which terminate favorably, recovery may be expected in from two to nine days, the shortest cases being those in which the pharynx alone is involved. The particular danger is from extension to the larynx, and death by suffocation or asphyxia.

Treatment.—Some cases seem to have been cut short by the local application of a sixty-grain solution of the nitrate of silver. The pain may be relieved by insufflations of morphine or the internal administration of bromide of potassium. In the early stages sucking of ice is useful for checking the inflammation, but later hot soothing inhalations impregnated with opiates or belladonna are more beneficial. Internally, large doses of quinine and tincture of iron, with alcoholics and nutritious foods, are important. If œdema is extensive, scarification should be practised, and, if the dyspnœa becomes urgent, tracheotomy must be resorted to, but, unfortunately, it is not usually successful.

Acute Rheumatic Sore Throat.—This is a painful affection of the pharynx and palate or tonsils, which occasionally extends to the larynx. It is usually of short duration, and is characterized by moderate congestion and swelling of the mucous membrane, which is attended by severe pain. It is most common in the subjects of rheumatism, and is not a very frequent disease in children.

Etiology.—The same as that of rheumatism in other parts of the body.

Symptomatology.—This affection is ushered in by sudden and severe pain in the throat, which continues for one or two days and then passes off with torticollis or other rheumatic pains. The temperature is elevated, the pulse is rapid, and very great pain is experienced in attempting to swallow the saliva or fluids. On examination of the throat, there is found more or less redness and swelling.

Diagnosis.—The affection is liable to be confounded with simple acute sore throat, from which it is to be distinguished mainly by the history of former attacks, by the peculiar character of the pain, which the patient himself will frequently recognize, and by the sudden metastasis to other regions at the end of the first or second day.

Prognosis.—The affection usually lasts from twenty-four to forty-eight hours. It is not in itself serious.

Treatment.—The salicylates, alkalies, and guaiacum are to be given internally. Sedative applications may be made to the throat when necessary to relieve pain.

Membranous Sore Throat.—*Synonymes.*—Croupous pharyngitis, Herpetic sore throat, Aphthous sore throat.

This is characterized by the formation of herpetic patches or blisters on the mucous membrane of the pharynx, palate, tongue, and cheeks, which eventually become covered with a fibrous exudate that forms into a pellicle or false membrane. It occurs more commonly in women and delicate children, but it may attack those apparently strong and in the best of health. It is observed at all seasons of the year, but is more frequent in the spring and fall, and is more prevalent in cold and damp climates than elsewhere.

Pathology and Pathological Anatomy.—Early in the disease ephemeral vesicles appear in the throat, which terminate within two or three days in resolution; or the vesicle may rupture and leave a small ulcer, which has a tendency to heal quickly; or several of these ulcers may coalesce and become covered with a fibrinous exudate having much the appearance of diphtheritic membrane. The tissues immediately about the ulcers are congested, swollen, and slightly elevated.

Etiology.—The affection usually seems to be caused by exposure to cold or to septic influences, or by the respiration of impure air. It is more frequent during epidemics of scarlatina and diphtheria, and therefore seems to be in some way due to the specific causes of these diseases.

Symptomatology.—Patients usually complain of *malaise* and ordinary sore throat for a day or two, or the affection may be ushered in with a decided chill. These symptoms are attended by smarting pain in the throat, heat of the skin, and high fever. The inflammation runs an acute course, sometimes extending to the orifices of the Eustachian tubes, and in other cases to the larynx. The ephemeral vesicles may appear in three or four successive crops. There are usually headache and loss of appetite. The tongue is furred, and the secretions from the mouth are viscid and have an offensive odor. Deglutition is generally exceedingly painful. Upon examining the throat, there may usually be seen several small vesicles about the size of a pin's head, filled with pus, about which there is a zone of congested and swollen mucous membrane. These vesicles may pass away without rupture, or, breaking, may leave small round ulcers which rapidly heal, or in other cases several of the ulcers may coalesce and become covered with a yellowish-white pultaceous exudate, which, when removed, leaves an excoriated surface which readily bleeds. The vesicles are frequently found on the pharynx, but more often on the palate and uvula, and the larger patches are often located on the tongue, on the mucous membrane of the cheeks, and sometimes on the tonsils. During the course of the disease herpetic patches usually appear on the lips, and membranous deposits form upon any ulcers which may happen to be present in other parts of the body.

Diagnosis.—The affection is liable to be mistaken only for diphtheria, from which it may be distinguished by the difference in the pseudo-membrane and in the constitutional disturbances. The membrane in this affection is of a yellowish-white color, thin, easily torn, and quite easily detached from the subjacent tissues, instead of having the grayish hue and the depth of the diphtheritic deposit, which involves the whole thickness of the mucous membrane and therefore cannot be easily removed. The presence of small vesicles or ulcers among the membranous patches is also an important sign in the diagnosis. The constitutional symptoms are much less pronounced in membranous sore throat than in diphtheria.

Prognosis.—The affection usually lasts from five to fourteen days. It is not dangerous *per se* except in children, in whom it sometimes extends to the larynx and causes death in the same manner as diphtheritic croup. However, it occasionally terminates in diphtheria, which may be serious. It may be followed by paralysis, even without the development of diphtheria. It occasionally attends syphilitic and tubercular sore throat.

Treatment.—The severe pain calls for the exhibition of anodynes, chief among which are opiates, bromide of potassium, and inhalations of hot-water vapor impregnated with benzoin, belladonna, or lupulin. Borax is sometimes grateful as a mouth-wash. The application of a sixty-grain solution of nitrate of silver to the patches sometimes allays pain and expedites recovery. But I have found the most relief from the application twice daily of a pigment composed of morphine, grs. v; carbolic acid, grs. xxx; tannic acid, grs. xxx; glycerin and water, each, ʒiv. Alum and other astringents are recommended, and are occasionally useful. The chlorate of potash has been recommended for this affection, but it usually causes severe pain and does not seem to be in any way beneficial. Antiseptic mouth-washes are useful; for this purpose a solution of permanganate of potassium, ten grains to the ounce, may be employed. The bowels should be kept open with saline laxatives; and tonics, such as arsenic, strychnine, and quinine, should be administered in appropriate doses.

Sore Throat of Small-Pox.—This is a pustular affection of the mucous membrane, similar to that affecting the skin. The eruption usually appears on the pharynx or palate before it is well marked upon the skin. It is attended by congestion, swelling, and pain. Occasionally there is profuse salivation. The pustules may be followed by deep ulcerations which extend down to the muscular tissues.

Treatment.—Soothing gargles and weak astringents are generally useful, but no special treatment can be recommended.

Sore Throat of Measles.—This is a catarrhal inflammation which constitutes only a part of a general inflammation of the respiratory mucous membranes; however, in some severe cases a fibrinous exudate is thrown out that gives it a diphtheritic character.

Etiology.—The same as that of the cutaneous eruption.

Symptomatology.—The throat is usually affected on the third or fourth day of the fever, several hours before the cutaneous eruption makes its appearance. The symptoms are those of a simple catarrhal inflammation, with a viscid mucous secretion, but not much swelling or pain. Occasionally diphtheritic patches make their appearance, but generally not until the ninth or tenth day. In this instance the pseudo-membrane is more friable than that of diphtheria and is not so uniformly distributed. Occasionally ulceration of the mucous membrane or abscesses have been observed.

Diagnosis.—The diagnosis may be readily made after the appearance of the cutaneous eruption.

Prognosis.—In those cases where the inflammation is simply catarrhal, resolution may be expected in seven or eight days. In those where diphtheritic deposits take place, the result is commonly fatal; four-fifths of these patients die.

Treatment.—The same treatment may be employed as that recommended for acute sore throat.

Sore Throat of Scarlet Fever.—This is one of the first and most constant manifestations of scarlatina, characterized in mild cases by simple congestion, in more severe cases by extensive swelling of the mucous membrane and glandular tissues, and in malignant cases by diphtheritic deposits.

Etiology.—The poison of scarlatina.

Symptomatology.—The affection is often ushered in by vomiting, with stiffness and soreness of the throat. The mucous membrane of the pharynx, palate, and fauces usually becomes congested several hours before the cutaneous eruption appears. In many cases the mucous membrane soon becomes swollen and the lymphatics enlarged. The inflammation often extends through the Eustachian tube to the middle ear, and may cause permanent deafness. In malignant cases there is at first great lividity of the mucous membrane, which soon becomes more or less covered with pultaceous deposits, which upon being removed leave excoriated surfaces. Abscesses or gangrene occur in some cases.

Diagnosis.—The principal points to be considered in the diagnosis are the sudden onset with vomiting, congestion of the mucous membrane, high fever, and the subsequent eruption and desquamation of the skin; and in some cases dropsy, which may develop at the end of two or three weeks.

Prognosis.—The affection may last from three or four days to several weeks. In the simple cases there is no danger from the throat-affection. In the anginous variety, where there is much swelling, about one-fourth of the patients will be lost. Of the diphtheritic variety about one-half prove fatal.

Treatment.—Local measures usually prove of little avail. Cohen recommends acidulated sprays, which he says are soothing. Emollients and poultices are commonly employed, with apparent benefit. The internal treatment is that indicated for the constitutional disease.

Acute Follicular Pharyngitis.—This is an acute inflammation of the pharynx, which expends its force mainly on the follicles. The disease is characterized by simple swelling and redness of the follicles in many cases, and in others by the formation of small vesicles on the pharynx, palate, and pillars of the fauces or tonsils, which to some observers have the appearance of an herpetic eruption. Indeed, Sir Morell Mackenzie treats of this affection under the title of herpetic pharyngitis, while by others it is considered as simply an acute sore throat. However, both of these terms have been applied to other affections described in this work, and, as the inflammation in this disease involves the same tissues that are affected in the universally recognized chronic follicular pharyngitis, we believe that the term we have selected is most appropriate. In this affection several follicles in the pharynx will be found swollen and red, and sometimes they will seem to be distended with secretions, giving them the appearance of small blisters or pustules. The pustules which are occasionally seen on other portions of the mucous membrane of the mouth usually rupture in a day or two and leave small round ulcers.

Etiology.—The affection is usually attributed to exposure to cold or to rheumatism; however, the inhalation of irritating substances, as dust, smoke, and gas, and the use of the voice in badly-ventilated rooms, have frequently seemed to cause the disease in adults.

Pathology and Pathological Anatomy.—The mucous membrane is swollen, the mouths of the follicles become stopped, and their pent-up secretions cause the pustular appearance.

Symptomatology.—In mild cases the patient complains of dryness or pricking sensations in the throat, which are commonly preceded for several hours by malaise. In severe cases there is much constitutional disturbance, with a hot skin, rapid pulse, and high temperature. There is usually a constant tendency to hawk and clear the throat, and if the disease extends to the larynx the voice becomes hoarse. Upon inspecting the throat, the mucous membrane is found red, and several follicles with a smooth, glistening surface, of an ovoid or hemispherical shape, and measuring from three to five millimetres in diameter, will be seen standing out about two millimetres from the surface. Often two or more of these will have coalesced back of the posterior pillars of the fauces so as to form longitudinal welts. In some cases the secretions will have collected in two or three of these so as to cause the appearance of pustules, which, later on, rupture, and leave small ulcers.

Diagnosis.—The affection is liable to be mistaken for simple acute sore throat, and in the pustular variety for membranous sore throat. The diagnosis in ordinary cases depends upon the peculiar prominence of the follicles and the circumscribed zones of inflammation about them. The pustular variety will be distinguished from membranous sore throat by the absence of large patches covered by false membrane.

Prognosis.—The disease usually runs from two days to one week. There is no danger to life, but there is a tendency to repeated attacks which may extend over several weeks or months.

Treatment.—The most satisfactory method of treatment is found in the administration of anti-rheumatic remedies and bitter tonics. Locally the application once a day of a spray of morphine, grs. v, carbolic acid and tannic acid, each, grs. xxx, glycerin and water, each, ʒiv, has given me the most satisfactory results.

Chronic Follicular Pharyngitis.—*Synonymes.*—Granular pharyngitis, Chronic catarrhal pharyngitis, Ulcerated sore throat, etc.

This is a chronic inflammation of the pharyngeal follicles and of the mucous membrane immediately surrounding them. It is most frequently met with in young adults from twenty-five to thirty-five years of age, but it is not uncommon in children. Mackenzie describes two forms of the disease,—the hypertrophic and the exudative. The former is often met with, but the latter is so rare that some authors even deny its existence. In the hypertrophic variety the follicles are enlarged and have an oval or hemispherical shape similar to that found in the acute inflammation. These vary in size from three to five or six millimetres in diameter and usually stand out about two millimetres from the surface. They are often of a yellowish-white hue, but at other times the mucous membrane covering them is of a deep-red color. In the exudative variety the follicles become filled with desiccated secretions, and have the same appearance as the follicles in chronic follicular tonsillitis, except that they are usually smaller.

Etiology.—Among adults the most frequent causes are tobacco-smoking, improper use of the voice in bad air, and repeated attacks of the acute disease; possibly, also, the use of spices and stimulants may act as a cause. Among children the most frequent cause is obstruction to nasal respiration, either by swelling of the turbinated bodies or by enlargement of Luschka's tonsil. Digestive disturbances are responsible for a considerable number of cases, and heredity seems to play some part in the etiology.

Pathology and Pathological Anatomy.—In the common variety the enlargements are made up principally of swollen epithelial cells; in the exudative, the swollen membrane stops the mouths of the follicles, which subsequently become filled with desiccated secretions. In either case the symptoms are largely dependent upon mechanical irritation caused by the enlarged follicles.

Symptomatology.—Ordinarily the general health is not impaired. Usually the first symptoms which attract the patient's attention are sensations of slight discomfort or stiffness in the throat, with at times unusual dryness or tickling and generally a frequent desire to hawk and clear the throat of mucus. Fatigue is frequently experienced after using the voice, and in some instances hoarseness is a common symptom. The senses of hearing and of taste are frequently obtunded. Sometimes the pricking sensations in the throat resemble those caused by a foreign body. Where the affection has extended to the larynx, patients are often obliged to stop and clear the throat before attempting to speak, and the voice may then be muffled or hoarse, or it may be natural for ordinary conversation and imperfect for singing. In children especially the voice may have a nasal intonation, due to obstructions in the naso-pharynx or nares. After hawking the patients expectorate small masses of thick mucus more or less tinged with dust, which gives it a blackish appearance. The tongue is generally coated; digestive disturbances are frequent and the bowels are usually constipated. In exceptional cases considerable difficulty is experienced in swallowing. Upon inspection of the throat the characteristic appearance already described will be noticed in some cases, but in others the whole pharynx is thickly studded with granulations, the furrows between which are of a lighter color, due to atrophy of the mucous membrane, and sometimes are filled with mucus which has a purulent appearance, due to the color of the membrane beneath. The disease often extends to the tonsils, and the base of the tongue and usually the larynx are more or less involved, presenting a slightly congested appearance, but little or no swelling. The pharyngeal veins are frequently enlarged, and often one or more may be seen running into and terminating in an enlarged follicle.

Diagnosis.—The diagnosis is not difficult unless ulceration has taken place. The latter condition, which is very rare, may be mistaken for syphilitic or tubercular sore throat. The simple and superficial character of the ulcers, together with the history, will enable one readily to distinguish it from the specific disease; and the same appearance, with absence of marked constitutional symptoms or severe pain, will distinguish it from tubercular sore throat.

Prognosis.—The affection, unless properly treated, generally lasts for several years, when it may gradually subside or terminate in atrophy of the mucous membranes, causing the affection known as *pharyngitis sicca.* Most cases of the hypertrophic variety may be cured in three or four months by appropriate treatment, at least so far as disagreeable sensations are concerned, but the voice may remain impaired for a long time. The exudative variety is peculiarly stubborn under the ordinary forms of treatment, but it may be readily cured by the galvano-cautery.

Treatment.—Our first attention should be directed to the digestive organs and the removal of all predisposing or exciting causes. Locally, astringent lozenges and sprays, which may be applied by the patient himself, constitute the best remedies. In those cases where the mucous membrane is very red and irritable, the application of iodoform in powder and of soothing alkaline sprays is found beneficial. In those where there are several follicles enlarged, but the mucous membrane is of a nearly normal color and there is little or no irritability, I have found the greatest benefit from the insufflation into the naso-pharynx, two or three times each week, of about two grains of a powder consisting of one part of muriate of hydrastin and three parts of pulverized acacia. The powder thus applied will gradually find its way downward, and thus keep up the local effect for several hours. In young children I have frequently seen very beneficial results from the internal administration of the syrup of the iodide of iron in appropriate doses, together with other tonics if indicated, such as quinine, arsenic, and strychnine. Cases which do not readily yield to this treatment may generally be cured by more radical local measures: these consist of destruction of the follicles by caustics. The simplest method is to incise each enlarged follicle and insert into the cut a pointed stick of nitrate of silver. This, however, is not always successful. Sir Morell Mackenzie recommends the application of London paste to one or two follicles at each sitting; others have used chromic acid for the same purpose; but by far the most satisfactory method, either in the hypertrophied or the exudative variety, is the direct application to the diseased follicle of the galvano-cautery. As soon as the wound thus produced has healed, the follicle will be found to have disappeared. Two or three follicles may be treated at each sitting, and subsequent applications may be made after healing has taken place, which will require from six to ten days. Where there are enlarged veins they should also be cut off with the galvano-cautery.

Scrofulous Sore Throat.—This is a disease of childhood which is characterized in the mild form by the physical appearances found in simple chronic sore throat, and in the severe form by ulcerations, which cannot readily be distinguished from those found in debilitated subjects, whether of tuberculous or of syphilitic origin. Cohen inclines to the opinion that this is a common sore throat in subjects of a latent inherited syphilitic taint.

Etiology.—Inherited syphilis, or a scrofulous diathesis.

Symptomatology.—The affection comes on insidiously, and gradually progresses, usually throughout a period of several months, until finally extensive ulceration takes place. The patient is in a debilitated condition, but the constitutional symptoms are not pronounced. With extensive ulceration there is generally a little pain; but often this symptom is absent. The ulcerative process progresses slowly, but may finally involve a large amount of tissue, so as to destroy a considerable portion of the mucous membrane of the pharynx or the soft palate.

Diagnosis.—This disease is likely to be mistaken for syphilis or tuberculosis only. There are no diagnostic symptoms or signs, but a consideration of the history and the constitutional symptoms as well as the local signs will generally enable the physician to arrive at a correct conclusion. This is a disease of childhood, whereas syphilis and tuberculosis are usually found only in more advanced age. Those cases in which there are simple congestion and swelling of the part cannot possibly be distinguished from chronic catarrhal sore throat, or from syphilitic sore throat in which there is no ulceration; however, these are of minor importance. When ulceration has taken place, the disease under consideration differs from syphilis in that there is no congested areola about the ulcer, the edges of which are slightly raised and everted, but not sharply cut or undermined as in syphilis. The discharge from these ulcers is slight, and they are much more slowly destructive than those of the specific disease. The strumous appearance of the subject is also a sign of importance. Tuberculous ulcers have no distinct line of demarcation, and are superficial; not so the scrofulous ulcer. In tuberculosis the fever, emaciation, and pulmonary signs differentiate it from the disease under consideration.

Prognosis.—These ulcerations are difficult to heal, but under appropriate treatment recovery may generally be expected.

Treatment.—The treatment which has been found most beneficial consists in the administration of tonics and nutritious diet, and the local application of alteratives and stimulants, daily at first and less frequently as healing progresses. For this purpose the tincture of iodine in full strength, or the sulphate of copper five to fifteen grains to the ounce of water, has been found most beneficial.

Acute Tubercular Sore Throat.—This is an acute affection of the throat, which runs a rapid course and is attended by the constitutional symptoms of tuberculosis. The affection is rare in children.

Pathology and Pathological Anatomy.—In the early stages it is characterized locally by gray granulations of small size beneath the epithelium. These granulations are usually grouped in patches. They bleed easily when touched, and are very abundant and prominent, closely resembling the mucous patches of syphilis, but lacking the inflammatory areola of the latter. The granulations are generally found on the palate, palatine folds, and pharynx, but later they may extend to the larynx.

Etiology.—The same as that of acute pulmonary tuberculosis.

Symptomatology.—The disease may begin primarily in the pharynx with symptoms of acute catarrhal inflammation, but in most cases the lungs are first involved. The patient suffers from intense pain, especially on attempted deglutition, in consequence of which rapid emaciation and loss of strength occur. The pain is of a sharp, lancinating character, and frequently extends to the ears. The pulse is rapid, and the fever persistent. The temperature ranges from 101° to 103–4° F., and in extreme cases reaches as high as 107°. The tongue is coated with a whitish fur, and the appetite is usually lost. On account of the weakness and pulmonary complications, dyspnœa is a prominent symptom. The patient is generally annoyed by a teasing cough, but in some instances there is none. The sputum comes mostly from the throat, and is not very abundant. Upon examination of the throat, the granulations already referred to may sometimes be seen in the beginning of the disease, but usually we find irregular, shallow, grayish ulcers, with indistinct borders; there may be one or more of these, or the ulcers may have coalesced and a large part of the palate or pharynx may be involved. An examination of the lungs will generally reveal the signs of pulmonary tuberculosis at the apex of one or both.

Diagnosis.—This affection is liable to be mistaken for syphilitic or scrofulous sore throat. The most important points in the diagnosis are the presence of the small granulations, acute pain, persistent high fever, and the signs of pulmonary disease. It may be distinguished from syphilitic sore throat by the intensity of the pain, by the persistent fever, and by the presence of small gray bleeding granulations instead of mucous patches, or by large, irregular, superficial ulcers instead of the deep ulcers with sharp-cut edges and inflammatory areola which are found in tertiary syphilis. This affection seldom occurs in children, but, when it does, may be distinguished from the scrofulous disease by the persistence of the fever, and by the superficial ulcer with no distinct line of demarcation, instead of the deep ulcer with sharply-defined edges; also by the pulmonary signs.

Prognosis.—The duration is generally from two to six months; many cases will prove fatal within six or eight weeks. Nearly, if not quite, all cases prove fatal.

Treatment.—The treatment is that suitable for acute pulmonary tuberculosis, in addition to which sedatives may be employed locally. Cocaine has been recommended for this purpose, but the relief which it gives is temporary and the constitutional effects are injurious. The most relief will be derived from the inhalations of steam impregnated with conium, belladonna, opium, or compound tincture of benzoin, or from the insufflation of powders containing morphine and bismuth. Iodoform has been highly recommended, but it is of doubtful utility.

Retropharyngeal Abscess.—This is a deep-seated inflammation of the pharynx, characterized by the formation of pus in the submucous tissues. It is most frequently met with in children, and it has even been observed in the new-born babe, but may also occur in adults.

Pathology and Pathological Anatomy.—The resulting abscess may be located in the naso-pharynx, the oro-pharynx, or in the laryngo-pharynx beyond the field of vision when unaided by the throat-mirror. It may be developed near the median line or upon either side. It is said to be confined to one side in about three-fourths of the cases. The lax attachment of the pharyngeal mucous membrane favors the formation of an abscess and allows matter to burrow easily in any direction, though it is inclined to gravitate downward. In some cases it has extended into the posterior mediastinum.

Etiology.—The affection is in most cases idiopathic, occurring most frequently in scrofulous children or in those suffering from inherited syphilitic taint. It sometimes results from scarlatina or from acute pharyngitis, erysipelas, or tonsillitis, but in adults it is more frequently the result of disease of the cervical vertebræ, and it is often of syphilitic origin. The exciting cause is usually exposure to cold or extreme warmth. Some cases follow wounds, as the swallowing of bones, pins, and other foreign substances. It has occasionally been produced by stricture of the œsophagus in consequence of the mechanical irritation attending forced deglutition.

Symptomatology.—The disease usually commences with deep-seated pain in the pharynx, and stiffness of the neck, followed by dysphagia and dyspnœa, and by hoarseness if the abscess is situated low. Usually in young children the first noticeable symptoms are dyspnœa and difficulty in swallowing, with stiffness of the neck, which causes the patient to keep the head in a certain position. Sometimes there are spasmodic attacks of dyspnœa resembling convulsions, and not unfrequently actual convulsions appear. In most cases the symptoms are obscure until the swelling becomes large enough to interfere with respiration or deglutition. According to Bokái, idiopathic abscess may develop in forty-eight hours, and secondary abscess in from seven to ten days; those proceeding from diseased bone are still more chronic in their course. Occasionally the disease is ushered in by a distinct chill, but generally there are only slightly chilly sensations and some headache, with but little fever. The pulse is usually weak and compressible. The pain is referred to the palate when the abscess is high up, but is commonly deep-seated and may extend over the entire throat. According to the location of the abscess, the head is usually thrown backward or to one side and maintained in that position. Tumefaction of the sides and front of the neck is frequently observed, and the parts may be painful on pressure. The difficulty in breathing will depend upon the location of the abscess. If in the naso-pharynx, it interferes with nasal respiration only. When located in the oro-pharynx, respiration is not greatly interfered with until it attains large size. If situated in the laryngo-pharynx, the pharyngeal mucous membrane is crowded forward over the larynx, and great dyspnœa results, which is subject to frequent exacerbations attended by stertorous breathing and occasional cough. When the abscess is located high up, the voice has a nasal twang. If it is situated low down, there may be hoarseness or complete loss of voice. If the abscess is large or encroaches greatly upon the oro- or laryngo-pharynx, deglutition, especially of solids, becomes difficult, and swallowing of liquids is frequently attended by the passage of a portion into the larynx, with consequent choking. Upon examination of the throat, if the abscess is located in the naso-pharynx, nothing may be seen; but ordinarily a tumid swelling, of a dusky-red color and semi-elastic doughy feel, will be found. Late in the affection, from the collection of pus the tumor may present at some point a yellowish appearance.

Diagnosis.—This affection is liable to be confounded with croup, œdema of the larynx, foreign bodies in the larynx, and cerebral or digestive disorders causing convulsions. The essential symptoms are the dysphagia, dyspnœa, altered voice, and pharyngeal swelling. From œdema of the larynx it may be distinguished by inspection, and by elevating the larynx, which procedure relieves the dyspnœa in the pharyngeal abscess, but does not in œdema. From croup it may be distinguished by the symptoms and signs: in croup the voice is lost, but generally it is not in this disease; in croup there is no swelling or dysphagia. From foreign bodies in the larynx it may be distinguished by the history and the signs upon inspection and palpation, together with the quality of the voice. In those cases attended by convulsions the diagnosis must be based upon the results of a careful examination of the parts.

Prognosis.—Idiopathic cases commonly terminate in from three to five days, and secondary cases in from seven to ten days. Most of these recover, though fatal results are not unfrequent. Cases following spondylitis may last from three weeks to several months, and a large percentage will finally prove fatal. In favorable cases the abscess usually opens spontaneously, unless sooner relieved, and with the escape of pus the symptoms at once subside. However, pus may burrow into the areolar tissue of the neck or into the ary-epiglottic folds and the pressure may cause suffocation; or with the bursting of the abscess this accident may result from pus escaping into the larynx. If the abscess burrows deeply into the mediastinum, it may open either into the œsophagus or into the pleural cavities: in either case a fatal result may occur. Death has resulted from the abscess burrowing behind the tonsil and perforating the internal carotid artery.

Treatment.—In the early portion of the attack, continual sucking of ice will sometimes abort the abscess; but when pus has once formed it must be evacuated as soon as possible. In making the incision care should be taken to avoid the internal carotid, by keeping as nearly as possible to the median line. As soon as the opening has been made, the patient's head should be thrown quickly forward, to prevent the passage of pus into the larynx. An ordinary bistoury, guarded to within a quarter of an inch of its point by being wrapped with adhesive plaster or a bit of cloth, is as good an instrument as any for making the incision. Subsequently tonic and supporting treatment should be adopted. The syrup of the iodide of iron is a most useful remedy, or the phosphates of iron and quinine may be given, or the syrup of the hypophosphites. Cod-liver oil is generally recommended, but it should be remembered that it is only a nutrient, and it is not necessary if the appetite is good. In the early part of these attacks the bromide of potassium, in doses of from three to five grains every three or four hours, should be administered to infants who show a tendency to convulsions

Anæsthesia of the Pharynx.—This is a rare condition, which I have not witnessed in childhood except as a result of diphtheritic paralysis. In adults it is more frequently caused by progressive bulbar paralysis.

Tonics, strychnine, and galvanism are appropriate remedies. In extreme cases feeding by the stomach-tube may be necessary so long as there is danger of food entering the air-passages.

Hyperæsthesia of the Pharynx.—This can hardly be said to exist as a disease, yet it is of frequent occurrence, as shown by the difficulty patients experience in allowing examination of the fauces or larynx. It is not uncommon in persons otherwise perfectly healthy. It is frequently found in children, but requires no special treatment.

Paræsthesia of the Pharynx.—This is of frequent occurrence in adults, but is not common in children. It is characterized by sensations of heat, pricking, swelling, weight, or of some foreign body in the throat.

Etiology.—It frequently follows the removal of foreign substances from the fauces, and then seems to result from the irritation or wound which they have produced. In some cases it is of purely hysterical origin. It is sometimes caused by a small ulcer, and is speedily relieved when the latter is cured. Occasionally it is due to varicose veins at the base of the tongue, or to enlargement of the glands, and may then be relieved by destruction of these with the galvano-cautery. In some cases no cause can be found, and it is then liable to be very obstinate and may continue for several months.

Treatment.—The most satisfactory treatment for this affection consists in daily spraying the throat with a solution of from fifteen to thirty grains each of carbolic acid and tannin to four drachms each of glycerin and water, together with the internal administration of quinine, arsenic, and strychnine in cases subject to neuralgia, or of iodide of potassium, salicylate of sodium, guaiacum, and similar remedies in subjects of a rheumatic diathesis.

Neuralgia of the Pharynx.—This is a rare condition, which I have not witnessed in children. If found, it should be treated on general principles: local applications of carbolic acid, aconite, or cocaine, in appropriate quantities, might prove beneficial.

Spasm of the Pharynx.—This is occasionally met with independently of paræsthesia or congestion of the parts, but it is usually caused by acute inflammations of the uvula or pharynx. It is sometimes associated with spasm of the œsophagus. I have not met with the disease in children. In the milder forms it is said to result from incomplete mastication. Lennox Browne says that "it may be distinguished from organic disease by the fact that the patient has difficulty, never amounting to inability, of deglutition quite irrespective of the consistence or temperature of the food." This sign, however, will not always hold good.

Inspection of the parts, digital examination, and the passage of the œsophageal bougie, together with the history, will enable one to make a correct diagnosis.

Prognosis.—The affection is tedious, sometimes lasting two or three years, but ultimate recovery may be expected.

Treatment.—Tonics, bromides, and the passage of œsophageal bougies have proved the most beneficial means of treatment.

Paralysis of the Pharynx.—There are four varieties of this affection: first, that occurring after diphtheria; second, that associated with facial paralysis; third, that associated with paralysis of the œsophageal muscles; and, fourth, that due to progressive bulbar paralysis. Of these the first is the only one that is likely to interest us in the treatment of children.

Paralysis of the pharynx is not an unfrequent sequela of diphtheria; it usually comes on in from two to four weeks after the beginning of the attack, though it has been observed earlier. It is characterized by some difficulty in swallowing, especially of fluids, and, on account of the paralysis of other muscles, by more or less difficulty in expectoration, and by the nasal timbre of the voice and an inability to articulate certain sounds, due to non-closure of the passage to the naso-pharynx. Thus, egg is pronounced *enk*, head *hent*, rub *rum*, etc. The pharyngeal affection is generally associated with more or less paralysis of the palate and of the œsophagus.

Symptomatology.—One of the most marked symptoms is that of difficulty in swallowing, which sometimes becomes so great as to necessitate feeding through the stomach-tube. The palate is seen to be relaxed, usually more on one side than on the other, caused by the tendency to unilateral affection of the muscles. Impairment of the special senses in some cases takes place, as of taste, smell, hearing, and vision. Owing to the involvement of the respiratory muscles, dyspnœa or even apnœa may occur, or paralysis extending to the cardiac nerves may lessen the pulsations to fifty or even forty per minute, or in other cases may greatly increase the frequency of the heart's action.

Prognosis.—The prognosis is grave when the respiratory or cardiac nerves become implicated, but if the paralysis is not marked, and if it is confined to the pharynx and palate, recovery usually takes place after three or four weeks, though it may be delayed for several months.

Treatment.—In the treatment of this condition tonics, especially strychnine and iron, are the appropriate remedies. The faradic current may be applied to the affected muscles with benefit in some cases, but usually its effects are not very satisfactory. In cases in which the food occasionally finds its way into the air-passages, the patient should be nourished through the stomach-tube or by enemas, in order to prevent the occurrence of pneumonia, which would be apt to follow the passage of food into the trachea.

Scalds and Burns of the Pharynx.—These are not very unfrequent accidents, especially among the children of the poor. The mouth, tongue, palate, nares, pharynx, œsophagus, larynx, and trachea are all affected in such cases. The accident most frequently occurs from the inhalation of steam, but sometimes from the inhalation of flame or hot air, as in burning buildings. It is speedily followed by great acceleration of the pulse, attended by fever, pain, inflammation, and swelling of the parts causing difficulty of deglutition, and dyspnœa caused by swelling of the larynx.

Diagnosis.—This is easy, on account of the history of the accident, the great pain in the part, and the appearance of the mucous membrane, which during the first few hours is of a whitish color, and subsequently is seen to be destroyed, uniformly or in patches.

Prognosis.—The prognosis is grave. Many patients will die within a few hours. If the patient survives beyond this period, the destroyed portions of mucous membrane slough, and profuse suppuration occurs, with very great exhaustion. The extension of the inflammation to the larynx is often the immediate cause of death. In some cases the immediate effects of the accident are recovered from, but the patient is left with chronic laryngitis which may be attended by stenosis of the larynx and trachea.

Treatment.—Immediately after the accident considerable relief may be obtained by the inhalation of anodyne vapors or by constantly sucking pieces of ice. Cold compresses or ice-bags applied about the neck are also useful in moderating the inflammation. If pain is severe, anodynes must be given internally or hypodermically. If dyspnœa becomes urgent, tracheotomy must be performed. Unfortunately, however, the operation will often fail to relieve the patient. Mucilaginous drinks, barley-water, rice-water, etc., may be given for nourishment and to allay the inflammation, if the patient can swallow; otherwise, food must be administered by enemata.

Foreign Bodies in the Pharynx.—Foreign bodies—such as pins, bristles, fish-bones, and too large pieces of food—frequently become lodged in the pharynx and give rise to great distress. In some cases large bodies press the epiglottis down upon the larynx and may cause suffocation and speedy death. Long, narrow objects are usually caught transversely, often as high up as the tonsils. Larger bodies are generally found resting upon the epiglottis or upon the larynx. Buttons, coins, etc., frequently slip into the valeculæ or pyriform sinuses.

Symptomatology.—Small bodies remaining in the pharynx usually give rise to pricking sensations which are especially noticeable during deglutition. They sometimes become very annoying, causing the patient to make constant efforts for their removal. Occasionally they give rise to extensive inflammation and swelling of the parts. Frequently, if the foreign body has itself been removed, the sensations which it caused remain for several days or even weeks or months.

Diagnosis.—There is some danger of mistaking foreign bodies in the pharynx for simple neuroses. The diagnosis will be based upon the history of the case, aided by careful inspection of the parts with a strong light and the laryngoscope. Frequently a swab of absorbent cotton will be necessary to remove the saliva before the parts can be examined. In some cases inspection will be rendered much easier by the application of a spray of cocaine, but care should be taken not to use this in too large quantity in young children.

Prognosis.—In many cases the foreign body will be dislodged by the patient's own efforts, within a short time; in others it may remain, causing severe inflammation and ulceration. Not unfrequently death results from the impaction of bodies of sufficient size to cause suffocation.

Treatment.—When the foreign substance can be seen, it should be removed by forceps. In some cases small substances may be readily removed by a swab of cotton passed well down to the opening of the œsophagus and drawn upward along the sides of the pharynx. Where the foreign body interferes greatly with respiration, unless it can be immediately removed tracheotomy should be performed at once.

Morbid Growths in the Pharynx.—Tumors of the pharynx are not of frequent occurrence, but papilloma, fibroma, fibro-sarcoma, sarcoma, adenoma, lipoma, osteoma, and cystoma have all been met with in adults. They are very rare in children. Papilloma and fibroma are much more frequent than other varieties. Tumors in the oro-pharynx usually grow upon one side near the tonsil, where they may be readily seen and removed; but when they occupy a lower site they may press upon the epiglottis or other portions of the larynx so as to interfere with respiration and articulation, or by their size may seriously interfere with deglutition.

Diagnosis.—This is usually readily made by a careful inspection of the parts.

Treatment.—Malignant growths are not likely to occur in children. The benign growths should be removed as soon as discovered. When located beneath the mucous membrane, a straight or crucial incision should be made over them and the tumor enucleated with the handle of the scalpel, the forceps, or the fingers. Pedunculated tumors may be removed with the steel-wire écraseur or the galvano-cautery. In case of urgent dyspnœa tracheotomy should be performed, to prevent suffocation, and the tumor subsequently removed.

Syphilitic Sore Throat in Children.—This is nearly if not quite always a congenital manifestation of the inherited disease, usually showing itself in the form of mucous patches on the membrane of the mouth, palate, palatine folds, tonsils, and sometimes the pharynx. It is frequently attended by an obstinate coryza, probably due to mucous patches on the Schneiderian membrane. The principal symptoms are those due to the coryza, which occludes the nasal passages, and in young children interferes with suckling. As the disease progresses, specific pustules, fissures, and ulcers are developed upon the mucous membrane of the nose, mouth, lips, and fauces. The pharynx is less frequently involved than the other parts.

The treatment is essentially the same as that for the adult. The child should be carefully clothed and the skin kept clean, and great care should be exercised as to its nutrition. A mercurial course at first seems absolutely necessary, but later in the disease the iodides are more efficient. Topical sprays or washes of alum, borax, or weak solutions of sulphate of zinc or sulphate of copper will often be required. The tincture of iodine or strong solutions of chloride of zinc or nitrate of silver may be found necessary in case of extensive ulcerations.

DISEASES OF THE TONSILS.

BY BEVERLEY ROBINSON, M.D.

CONSIDERATIONS OF ANATOMY, PHYSIOLOGY, AND PATHOLOGY.

To the remarkable grouping of glands and follicles in the mucous membrane at the side of the base of the tongue, in an excavation limited by the two pillars of the fauces, we give the name of tonsils. An analogous collection is found also at the vault of the pharynx. If we examine the tonsils in a series of animals, these organs are seen to present numerous differences as to their size and configuration. Among reptiles, they exist only in crocodiles; in rapacious birds, large follicles behind the orifices of the Eustachian tubes have been described under this name. As regards the tonsils of the mammifers, they form either a simple sac with a single orifice, or horizontal leaves with small openings, or a large number of short, branched canals whose openings are scattered without order.[1]

In man the tonsillary region presents considerable obliquity anteroposteriorly and from the outside towards the interior. It is this obliquity, according to Richet,[2] which allows the surgeon to see this region readily when the jaws are opened widely; the tonsil is made more prominent by the tension of the posterior faucial pillar. The tonsils are variable in size. They are oval or almond-shaped bodies, flattened transversely, situated one on either side and projecting slightly into the isthmus of the fauces. Each tonsil is about twelve millimetres long and eight millimetres wide, and the thickness equals the width. Sometimes the tonsil is nearly absent; again it is so large as to force the pillars of the fauces out of their usual position and make a mass of some size in front of the pharynx.

It is evident, then, that the size of the tonsils may vary greatly and yet be considered normal. Accordingly, when we inspect the fauces, especially in children, we should not pronounce an enlargement morbid unless we discover certain symptoms of an annoying or painful nature obviously dependent on this increase in dimensions. According to Lennox Browne,[3] the tonsils when normal should not protrude beyond the plane of the anterior pillars. Still, size is not the only thing to be considered, since it is not uncommon to meet with tonsils which are diseased and at the same time very small,—*e.g.*, some tonsils containing cheesy masses in their lacunæ. On the other hand, they may be considerably larger than usual, and yet occasion no morbid symptoms.

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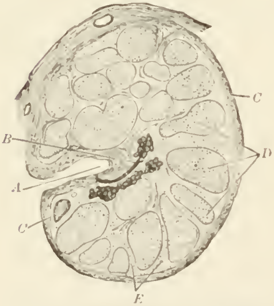
[1] Stannius and Siebold, Dictionnaire encyclopédique des Sciences médicales, vol. iv. p. 16.

[2] Dictionnaire de Médecine et de Chirurgie, vol. ii. p. 112.

[3] The Throat and its Diseases, 2d ed., p. 57.

An increase in the size of the gland always takes an inward direction, on account of the resistance to its development exteriorly of the muscular layers upon which it lies. The tonsils are composed of a considerable number of follicles, compound in character, whose ducts open into one another and terminate in twelve or more orifices of variable form. These last are visible on the surface of the tonsil, and mark the entrances to the crypts or lacunæ. According to Delavan,[1] who has made some original and very interesting researches in this line, the crypts of largest calibre contain "a yellowish substance composed of fat-molecules, loosened pavement epithelium, lymph-corpuscles, small molecular granules, and cholesterine-crystals." It is in the bottom of these lacunæ that those cheesy masses form which are so offensive in certain inflammations of the gland and which may in time be transformed into calculi. When the orifices of the mucous follicles are small, the surface of the tonsil is smooth and even; but this condition is relatively infrequent, and the usual appearance is that of a surface with numerous indentations. In the spaces between the crypts are a number of closed lymphatic glands, embedded in the connective tissue. The surface of the tonsil is covered with pavement epithelium, which as well as the mucous membrane (a continuation of the buccal membrane) extends by numerous prolongations into the flask-like cavities of the different lacunæ.

Fig. 1.



Section of the Healthy Tonsil (Mackenzie).—*A*, hilus; *B*, mucous gland; *C*, epithelial covering; *D*, lymphatic follicles; *E*, stroma.

Surrounding the tonsil is a vascular connective tissue, in which are included a large number of closed follicles containing numerous cells and free nuclei surrounded by a clear fluid. The tonsils are in relation exteriorly with the superior constrictors of the pharynx and the internal pterygoid muscles, and lie opposite the angle of the jaw; or, more correctly stated, the centre of the tonsil corresponds with the posterior alveolar foramen (Mackenzie). This circumstance explains the acute pain caused by pressure upon the angle of the jaw when the tonsil is inflamed. The tonsil can be explored through the soft part of the subhyoidean region. By combined external and internal pressure, hypertrophy or the presence of an abscess or a cyst may be determined. Between the tonsil and the pterygoid muscles there is a mass of fatty tissue which is continuous with that in the neck. A tonsillar inflammation may give rise to a phlegmonous process in this tissue, which may extend as far down as the clavicle; or an abscess of the tonsil may open into this tissue and the pus work its way downward to the same point.

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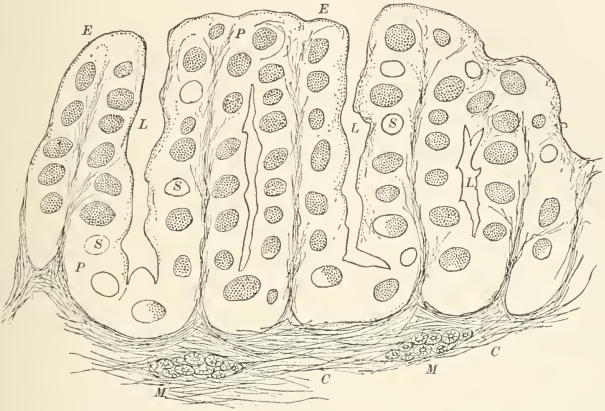
[1] Archives of Laryngology, vol. i. p. 337.

Fig. 2.



Section of a (Slightly) Hypertrophied Tonsil (Browne).—*E*. stratified epithelium covering the surface and lining; *L*, lacunæ or crypts; *P*, parenchyma, the adenoid tissue of which is not shown; *F*, lymph-follicles; *S*, lymph-spaces, mechanically voided of their contents, in section; *M*, mucous glands; *C*, capsule, or peritonsillar membrane, trabeculæ of which pass into parenchyma.

Behind the muscles lie the external and internal carotid arteries, the internal jugular veins, and the pneumogastric and glosso-pharyngeal nerves. According to Chassaignac, in old people the internal carotid is apt to bend towards the tonsil, so that its convexity is not far removed from the deep surface of that gland. As this disposition is not present in children, it is plain how unlikely an operator is to wound the artery in making an incision of the tonsil or opening an abscess. Burns, Portal, and Bellard report cases where injury was done to the carotid artery. The condition in which it would be necessary to locate the vessel carefully would be where a tumor was to be extirpated which protruded into the sterno-mastoid region.

The tonsil does not always preserve its position in the tonsillary excavation. On the contrary, especially when enlarged, it may descend along the lateral wall of the pharynx, so that the surgeon is obliged, either with finger or with instruments, to search deeply in order to reach its lower border. Under these circumstances we are compelled, if we would see the whole of it, to press the tongue well down with the spatula.

The tonsils are either sessile or pedunculated. The latter disposition is, of course, favorable to extirpation by means of a snare. When the tonsils lie down in the pharynx, it is difficult to appreciate the trouble they cause, and it is likewise not easy to excise them with the guillotine. The tonsils are placed more or less deeply in the tonsillar excavation. When they become hypertrophied and extend beyond the pillars of the fauces, they are apt to become engorged by pressure or the constriction of the pillars. This constriction is increased by the use of astringent applications. We can thus understand how very frequently these applications do more harm than good. When the gland has not grown so as to extend beyond the pillars of the fauces, astringent applications are almost always useful. Sometimes to the touch tonsils feel quite large, and yet when we introduce a tonsillotome they get away from the grasp of the instrument. This is due solely to the active contraction of the pillars, which become constricted and include the gland between them.

The large arterial trunks are from one-half to four-fifths of an inch from the surface of the tonsils. The tonsillar branch of the facial artery (a branch of the external carotid) is often quite large, and when cut sometimes gives rise to serious and even alarming hemorrhage. According to Zuckerkandl,[1] the tonsillar artery, in traversing the tonsillar capsule, forms adhesions with it which hold it open and prevent its retraction when it is cut through. Hence the necessity when excision is made of not going beyond the parenchyma of the gland: partial extirpation is, therefore, a much safer operation than complete ablation. The arterial supply of the tonsil is abundant, and in proportion to the size of the gland. It comes from the inferior pharyngeal and the two palatine arteries, superior and inferior. The external surface of the tonsil is covered by a venous plexus which receives the little veins which come out of the gland, and is continuous posteriorly with the pharyngeal plexus of veins.

The lymphatic vessels of the tonsil go to the lymphatic ganglia at the angle of the jaw. It is not infrequent to recognize an adenitis at the angle of the jaw as a result of amygdalitis. The nerve-supply is from the fifth pair and from the glosso-pharyngeal nerve (Bosworth). According to Pappenheim, it terminates by fine arborizations in the mucous membrane.

It is generally admitted that the function of the tonsil is twofold. In the first place, the numerous acinous glands secrete a considerable amount of clear, viscid liquid, much like that secreted by the small buccal glands. It is destined to lubricate the alimentary bolus and to facilitate its passage through the isthmus of the fauces and in its descent to and along the œsophagus. The lacunæ are somewhat like reservoirs which always contain fluid and which send forth their contents into the buccal cavity when the superior constrictor muscles, the palato-glossus, and the palato-pharyngeus press upon them as they contract in the effort of deglutition. This fluid is seen under the microscope to contain pavement and nuclear epithelium, and occasionally some leucocytes, crystals of cholesterine, and perhaps micrococci. It is these solid elements which, under certain conditions already referred to, go to form the solid caseous masses with fetid odor which we frequently meet with half extruded from the tonsillar crypts.

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[1] Zur Frage der Blutung nach Tonsillotomie, Wiener Med. Jahrbücher, Heft vi., pp. 309–327, 1887; Centralblatt für Laryngologie, etc., 1887, p. 214.

In the second place, in consequence of the existence of numerous closed follicles in the deep layers of the tonsil, these glands resemble, according to Brucke, other ductless or blood glands, like the lymphatic ganglia, the spleen, the thymus, etc. Liégeois[1] also, in his thesis, classified them in a similar manner. There are other resemblances, however, which may be summarized as follows: 1. The tonsils often become hypertrophied. 2. This hypertrophy may coincide with general hypertrophy of the lymphatic ganglia. 3. The closed follicles, as well as the tonsils themselves, are larger proportionately in children than in adults: it seems probable, therefore, that their function at this period of life is relatively more important. Further, it may be inferred that these glands serve a purpose in the economy similar to that of glands analogous in structure,—*i.e.*, they modify notably some of the constituents of the blood, and particularly they aid in the formation of the white corpuscles. They also contribute to the elaboration of the lymph, the principal formative and regenerating constituent of the blood (Saint-Germain). Nevertheless it is doubtful whether the ablation of the tonsils could lead to emaciation (Headland) or other troubles of general nutrition, since they are, from a functional point of view, merely adjuncts of other organs. The enlargement of the tonsils for which excision is performed should rather be regarded as an expression of a previous general dyscrasia. There are unquestionably pronounced physiological relations between the tonsils and the organs of generation (Harvey and Crisp, Verneuil, P. James). As regards their pathology, it may be added that local evidences of the specific dyscrasiæ are frequently seen on the tonsils.

TONSILLITIS.

In general, the tonsils are prone to become affected by the same pathological changes as other lymphatic structures. In children these changes are almost always limited, when we consider merely the tonsil itself, to acute or chronic inflammation. The former may be superficial and catarrhal in type, terminating usually in resolution; or it may be deep-seated and parenchymatous. Infrequently in these cases, and then only in later childhood or towards puberty, do tonsils affected with acute inflammation go on to the stage of suppuration. In fact, I cannot recall a single instance in which I have seen suppurative tonsillitis in a small child.

Chronic inflammation of the tonsils is in the vast majority of cases accompanied by more or less hypertrophy or enlargement of the gland. There are occasional instances, however, in which the tonsils are affected with chronic inflammation without being at all enlarged, but, on the contrary, are under the usual size. In these cases it is not infrequent to discover fetid cheesy formations in one or more of the distended lacunæ. Sometimes these masses will come forth spontaneously from the tonsil, but frequently we are obliged to exert moderate pressure on the sides of the lacunæ so as to force them out. Now and then I have been compelled to make a superficial incision through the mucous membrane covering the tonsil in order to reach them and scoop them out with a small spoon. It is common to find with acute or chronic inflammation of the tonsils more or less inflammation, analogous in kind, affecting the pharynx, uvula, palate, and isthmus of the fauces. Such conditions, when well marked and when the tonsillar inflammation is very slight, or indeed absent, are described as pharyngitis, angina simplex, chronic relaxed throat, etc. Frequently these conditions, especially the acute forms, are merely symptomatic of general diseases, such as erysipelas, eruptive fevers, virulent diseases, etc. Tonsillitis which is characterized by a tough, adherent, membranous exudation, and is a mere localization of diphtheria, we do not here consider. Follicular or lacunar tonsillitis we shall deal with farther on. There is a form of tonsillitis, graphically described by Rilliet and Barthez,[1] Da Costa,[2] and a few other writers, under the name of herpetic or ulcero-membranous tonsillitis, which may be confounded with either of the two preceding forms. It usually begins, according to the writers referred to, with an eruption of herpetic vesicles on the tonsil. Soon these vesicles rupture, and the superficial surface of the tonsil is englobed or covered with a membranous envelope, less adherent than the membrane peculiar to true diphtheritic angina, and more continuous than the whiter, cheesy exudation of lacunar tonsillitis. I do not remember to have recognized this form of disease in its initial or vesicular stage, and I have but rarely encountered a tonsillar disease which corresponded in its symptoms and progress with the later stages of the affection, to descriptions of which the reader has been referred.[3] Follicular tonsillitis is often an accompaniment of acute parenchymatous tonsillitis; herpetic tonsillitis is said more frequently to complicate superficial or erythematous tonsillitis. Although some authors make a separate variety of acute tonsillitis in instances in which the rheumatic poison has apparently acted as a constitutional cause of the local inflammation, this seems to me to be undesirable from a nosological point of view, for we might, in a similar manner, extend the number of varieties of the disease almost indefinitely.

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[1] Anatomie et Physiologie des Glandes vasculaires sanguines, Thèse d'Agrégation, Paris, 1860.

ACUTE TONSILLITIS.

Definition.—An acute inflammation of the tonsil or tonsils, which may be superficial or parenchymatous, and may terminate in resolution, suppuration, or chronic enlargement.

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[1] Traité clinique et pratique des Maladies des Enfants, vol. ii. p. 201, Paris, 1887.

[2] Amer. Jour. Med. Sci., July, 1870, p. 129.

[3] Da Costa appears to regard herpetic and ulcero-membranous angina as the same disease; but Rilliet and Barthez make of them two distinct diseases.

Synonymes.—Quinsy, Amygdalitis, Inflammation of the tonsils; Latin, Inflammatio tonsillarum; French, Amygdalite; German, Entzündung der Mandeln; Italian, Angina tonsillare.

History.—Tonsillitis was described by Hippocrates. In modern times Sauvages, Cullen, Louis, and others have contributed many points of interest in the history of the disease. The most complete descriptions of this affection written within the past twenty-five years may be found in the French Encyclopædia of Medicine, the French Dictionary of Practical Medicine, and the Manual of Diseases of the Throat by Sir Morell Mackenzie.

Etiology.—Of the predisposing influences the most important are—

1. *Age.*—Tonsillitis is very rare in infancy.[1] There is, however, at this age an appearance of redness and fulness in the tonsils that is normal, and is occasionally confounded with inflammation. In childhood, youth, and especially at the age of puberty, tonsillar inflammation is quite frequent, although even then it is not so commonly met with as in early adult life. It is somewhat remarkable to note the fact that acute inflammation of the tonsils is so unusual in infancy, when we remember that a large proportion of cases of enlarged tonsils are met with during the first few years of life. According to Sir Morell Mackenzie,[2] the precise percentage is twenty-six and one-half.

2. *Sex.*—The number of boys and of girls attacked is probably about equal, although I have seen more boys with acute tonsillitis, owing to the fact of their greater exposure, as a rule, to changes of temperature and to other accidental conditions which are likely to occasion an attack of this disease,—*i.e.*, wet feet, sudden arrest of perspiration after athletic games, etc. This is particularly true of boys in the lower classes of society, who are less carefully guarded by their mothers.

3. *Temperament.*—Pale, lymphatic girls and boys are the most apt to have tonsillar inflammation. Whenever the strumous constitution is well marked, the slightest accidental causes are sufficient to produce this effect. Recurrences of tonsillitis are frequent in those who inherit gout or rheumatism, and it is not unusual even in children to trace an evident connection between this local inflammation and other manifestations of a rheumatic constitution.[3] In certain families, independently of the existence of an evident dyscrasia peculiar to gout or rheumatism, there appears to be a marked hereditary tendency to inflammation or hypertrophy of the tonsils. One or more attacks of tonsillitis make the patient more liable to a recurrence of this inflammation. The existence of a certain amount of enlargement or hypertrophy seems to make the little patients peculiarly susceptible

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[1] Reid reports a case of suppurative tonsillitis in an infant only seven months of age (Archives of Laryngology, vol. i. p. 229).

[2] Loc. cit., vol. i. p. 49.

[3] Dict. de Méd. et de Chirurg. prat., Paris, 1865, vol. ii. pp. 118, 449; Lennox Browne, The Throat and its Diseases, 2d ed., p. 225; C. Haigh Brown, Tonsillitis in Adolescents, London, 1886.

to a return of the inflammation. Under these circumstances the slightest disturbance of the stomach or bowels, or any undue exposure to cold, high winds, or to dampness, is very apt to be followed by sore throat. There is no doubt that tonsillitis is more general whenever rapid changes of temperature occur, such as are common in our variable climate during the spring and autumn. It is also a well-observed fact that attacks of tonsillitis in children are more frequent in those seasons when measles, scarlatina, and diphtheria have been unusually prevalent. The epidemics of tonsillitis formerly described by different authors were probably connected to a large extent with the causes which produced the foregoing diseases. It is at least doubtful to what extent they should be considered as outbreaks of a separate and distinct affection.

It is perhaps true to a certain extent that special local conditions are important as a predisposing cause. It is also probable that certain atmospheric conditions, so much insisted upon by older writers, have been the cause of epidemics the precise nature of which remains doubtful to us of the present day. Among these I would cite the epidemic, reported by Dr. Mayene, which prevailed at Gordon, in France, in the autumn of 1818. In this epidemic, simple inflammatory tonsillitis, with or without fever, lasting from four to six days and terminating in abscess or resolution, attacked almost exclusively children and young people under eighteen years of age.[1]

Among the exciting causes, the influence of cold and humidity acting locally upon the neck or feet is very generally recognized by writers as an efficient cause of tonsillitis. Sitting in a draught when wet or perspiring, and neglect in changing wet clothes of any kind, are frequent causes of tonsillitis. It is perhaps a less familiar fact that exposure to an overheated and vitiated atmosphere will also occasion the development of tonsillitis in susceptible children. Septic causes of tonsillitis are also often met with, and I am constantly advised of the fact that defective drainage and drinking impure water may give rise to recurrent attacks of acute tonsillitis in children. In many of these cases, according to Kingston Fox[2] and Browne, the differential diagnosis from acute tonsillitis due to cold or other causes is made by the fact that the septic cases are bilateral in the beginning. No doubt some children are rendered more susceptible to septic causes by the presence at the same time of a rheumatic habit or a strumous constitution. Nevertheless, whenever recurrent attacks of tonsillitis occur in a child, or among children in the same family, it becomes a duty to see to it that the basins, sinks, lavatories, drains, and pipes shall be examined as carefully as possible, to discover if there be any defect in the plumbing through which sewer-gas may gain entrance to the house.[3]

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[1] Dict. encycl. des Sciences médicales, vol. iv. p. 19.

[2] Trans. of the Med. Soc. of London, vol. ix. p. 255.

[3] Goodhart, Diseases of Children, Amer. ed., 1885, p. 106. It is generally known that, by pouring oil of peppermint into the pipe leading to the roof and connecting with the main drain of the house and thence to the soil-pipe, if any notable opening exist in the

The inflammation in tonsillitis usually implicates the pillars of the fauces and soft palate. Traumatism or the inhalation of irritating vapors or gases may act as occasional direct causes of tonsillitis. Again, the swallowing of chemically acrid substances, or the impaction of some foreign body in the tonsil,—such as a fish-bone, a piece of oyster-shell, the bristle from a tooth-brush, a nut-shell, pins, or needles,—may cause an irritation or wound of the tonsil in the act of deglutition, and thus excite inflammation of this gland. Such causes of inflammation belong to any age, but are more likely to occur in childhood, when inattention to the accidental circumstances of disease is so marked. In children the cheesy formation in the crypts or lacunæ is at times a cause of recurrent tonsillitis, acting, as I believe, mechanically in producing this effect. I have never seen true calcareous tonsillar formations in children. At times it is really difficult to determine a reason for the development of the tonsillar inflammation, and it is then fair to assume that certain processes are carried on in the system by a perversion of which the poison that occasions an outbreak of tonsillitis may be developed.

Symptoms.—These differ according to the form of the disease which is present. We shall therefore, for the sake of clearness, describe three varieties: 1. Acute superficial or erythematous tonsillitis. 2. Acute follicular or lacunar tonsillitis. 3. Deep or parenchymatous tonsillitis.

1. *Acute Superficial Tonsillitis.*—After sudden or prolonged exposure to cold or wet, or in consequence of improper food and overheated, vitiated atmosphere, a healthy child complains more or less of weariness and general malaise. It seems drooping and out of sorts. Frequently there are headache, nausea or vomiting, chilly sensations, and some elevation of temperature. The bilious condition may be, indeed, very marked, and stomachal disturbance may last during several days. Simultaneously with these initial symptoms, or a little later, the child complains of slight heat or pain in the throat and difficulty of deglutition. The pain, at first perceived only during deglutition, later becomes permanent and increases in intensity. Frequently it radiates towards the angle of the lower jaw. Here there is often slight swelling of the lymphatic ganglia. Pressure exerted in this region augments the pain and indicates also the side where inflammation particularly exists. Sometimes, owing to pressure on the posterior palatine fold, pain in the ear is complained of. This is explained by the attachments and course of the staphylo-salpingeus muscle, which goes from the soft palate to the pharyngeal extremity of the Eustachian tube. Pain may also be due to simple irritation of the chorda tympani. Whenever, by reason of the pressure exerted upon the Eustachian tube, or on account of an extension of the inflammation, the little patients complain of noises in the ears, or have evidently impaired hearing, we should examine the ears with

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pipes or their joints, it will be almost immediately revealed by the odor of peppermint in some part of the house.

the otoscope and guard against avoidable sequelæ, as these symptoms point to inflammation of the middle ear, and are often premonitory of an impending suppuration and perforation of the drum.[1] When there is marked swelling of the tonsils, the voice assumes a characteristic nasal intonation. There is often occasional cough, with frequent painful expectoration of viscous and stringy mucus which collects in the throat. If the child be very young, he usually swallows this mucus. There are thirst, inappetence, and lassitude. The breath is usually foul, the tongue coated, the bowels constipated. The urine is small in amount, high-colored, and loaded with urates. The breathing is accelerated, the pulse rapid and full. The temperature rises rapidly, and in a few hours may reach 102° or 103° F. The pulse ranges from one hundred and ten to one hundred and thirty per minute.

If at this time the throat be examined with the aid of a tongue-depressor and before a good light (sunlight or artificial light), the tonsil (or tonsils)[2] will be found red and swollen. At first the affected surfaces appear somewhat dry and glistening; later they are covered by a certain amount of grayish exudation, which lies here and there and is slightly adherent. It is composed mainly of mucus, epithelial cells, pus-cells, and serum.[3] It may be readily detached from the inflamed gland by gargling, or by brushing lightly with a camel's-hair brush. Accompanying the tonsillar inflammation we usually notice slight redness and swelling of the uvula, soft palate, and pillars of the fauces. Pharyngitis, properly speaking, is infrequent unless we have considerable general inflammation of the throat.

At first, on account of dryness of the throat, there are frequent efforts at deglutition. Later on, when the act of swallowing becomes more painful, cooling drinks are usually the only form of nutriment which the child will take willingly. These often seem to afford temporary relief to the pain in the throat. The rise of temperature is ordinarily proportionate to the amount of inflammatory pain and swelling in the throat. The latter symptoms are more marked, as a rule, in the first attack than they are in the subsequent ones. In some few exceptional cases the inflammation is purely local, and gives rise to no general symptoms whatever,—the only symptoms which direct attention to the child's throat being the complaint of pain in this region and the evident, though slight, difficulty in swallowing. The child, if of a nervous temperament, is often very restless, sleeps fretfully, and at night, if the inflammation be severe enough to cause much fever, may be somewhat delirious. Other children, on the contrary, remain in a very quiescent condition, seemingly overpowered by the disease. Insufficient and careless observation might make of this a state of adynamia, in view especially of the dryness of the tongue which is present, and which is due to breathing through the half-open mouth. Two or three days will ordinarily suffice to show the fallacy of this judgment. When cough exists, which is rarely,[1] it is caused by the inability of the little patient to expectorate or swallow the whole of the viscid mucous secretions which collect in his throat and which in part may find entrance into the larynx. It may also be caused by the titillation of the base of the tongue or of the epiglottis by the elongated, relaxed uvula.

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[1] J. Solis-Cohen, article in Pepper's System of Medicine, vol. ii. p. 381.

[2] According to Sir Morell Mackenzie (Diseases of the Throat, vol. i. p. 52), "the inflammation is generally limited to one tonsil." Cohen, in Pepper's System of Medicine, also writes, "Occasionally both tonsils are involved simultaneously, but this is far less frequent than involvement of the second tonsil a few days later."

[3] Flint, Practice of Medicine, 6th ed., p. 396.

2. *Acute follicular or lacunar tonsillitis* is a disease frequently met with among children, and its clinical importance is considerable. In this disease the inflammation affects not merely the mucous membrane covering the surface of the tonsils, but also that lining the interior of the lacunæ or crypts. Upon examination of the tonsils, we notice at first, in the mouths of the crypts and extending into their interior, a number of small, white, pultaceous, cheesy-looking masses. These masses are more prominent than the membraniform layer of diphtheria. They are also more easily detached, and beneath them there is really no evidence whatever of ulceration or abrasion of tissue. Examined under the microscope, they are composed of mucus, pus-cells, epithelial cells, serum, and numerous bacteria. They are frequently called by patients themselves, and, I regret to add, by some distinguished authors, ulcerations. They are, however, not ulcerations. It is true that there is a form of disease, very rare relatively in children, to which we may properly give the name of ulcerous tonsillitis. In this disease a small layer of mucus and pus-cells forms directly under the surface-membrane of the tonsil. This membrane comes away after a few days, leaving behind it a superficial erosion. The mouths of the crypts, also, are often red and slightly eroded.[2] I must confess that I have never met with cases which Sir Morell Mackenzie describes as "honeycombed with ragged and indolent ulcerations."[3]

In follicular tonsillitis the constitutional symptoms are often very severe. The chilly sensations, headache, anorexia, insomnia, and other symptoms which invariably attend a marked febrile state are usually present. The fever itself in the first twenty-four hours may often reach 104° or 105° F., and make us—until we become familiar with such cases—solicitous as to the result. Fortunately, the prognosis is always good, and usually in four or five days the temperature subsides and the other attendant symptoms referred to disappear with it. It is no uncommon circumstance, however, for a notable degree of general depression of the system to persist for many days after the disappearance of all local symptoms.

The main source of anxiety in follicular tonsillitis is the possibility of confounding it with diphtheria, from which we shall endeavor further to distinguish it. Whilst it is true that many cases of follicular tonsillitis cannot be definitely and distinctly traced to a septic cause, I am more and more convinced that the influence which underlies such evident derangements of the *prima via*, accompanied by chills and anorexia, is due to specific germs or entities present in the body. If we do not find them, I prefer to think that our means of research are not capable of demonstrating them. The symptoms which in children accompany all but the mildest forms of acute simple tonsillitis are of too distinctly grave and serious a nature for us to doubt the presence of some poisonous substance in the body at large. The resemblance between the symptoms of this disease and those of other diseases whose pathogenic micro-organism is known, leads me to conclude from analogy that a more or less similar germ is present here also.

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[1] Browne states, in regard to cough, that there is "none."

[2] See Dict. encycl., tome iv. p. 20.

[3] Loc. cit., p. 52.

According to Sir Morell Mackenzie,[1] the constitutional phenomena are less marked in the follicular form of tonsillitis than they are in an attack of quinsy. This statement I have not been able to corroborate; but when Dr. Mackenzie states, a few lines farther on, that "in follicular tonsillitis the swelling of the tonsils is less considerable," I am wholly in accord with him. No doubt Dr. Mackenzie had in view, when he wrote his graphic account of tonsillitis, what takes place habitually in adults rather than in young children. But it is especially in our third variety of tonsillitis that the differences are more apparent and should be most emphasized.

3. *Parenchymatous tonsillitis*, or acute inflammation of the substance of the tonsil, is not a frequent disease of childhood, if the standard of comparison be the disease as it occurs in adult age. In the early period of life, and in youth, the rule is for a case of this kind to tend towards resolution. During early manhood or in middle life, if this disease occurs it tends in many instances towards suppuration or the formation of an abscess in the gland or in the peri-tonsillar cellular tissue. Occasionally, it is true, from the history of the case, or from the appearance of the tonsil after this form of tonsillitis is said to have occurred, it would seem that undoubtedly there must have been acute suppuration. Dr. Goodhart[2] reports such an instance in a girl six years of age, who, when she came under treatment, showed a large, deep ulcer on the left tonsil, "which could," he thinks, "only have originated in acute suppuration of the tonsil." Still, judging carefully from my own experience (and that of my colleagues) in throat diseases, I am compelled to state that suppurative tonsillitis in childhood, up to the age of ten to fifteen years, is a very uncommon disease. According to Vidal,[3] suppurative tonsillitis is rather a *special form* of tonsillitis than one of the ordinary terminations of this disease. Otherwise, parenchymatous inflammation of the tonsils in children presents many of the symptoms witnessed in adults, although usually of a more moderate sort.

One or both tonsils may be attacked. The tonsils are much more enlarged than they are in the preceding varieties of disease which we have described. The inflammation extends to the surrounding soft parts, also, in a more marked degree. The palate, uvula, pillars of the fauces, and even the pharynx, may all become very red, swollen, and slightly œdematous. The uvula, particularly, is often greatly tumefied, elongated, and œdematous, assuming the aspect at times of a sac filled with jelly: sometimes it adheres by viscid secretion to the swollen tonsil on either side. The submaxillary gland becomes engorged and sensitive. The tonsils themselves are sometimes so much swollen that with their coverings they fill up a large portion of the pharyngeal cavity and are very closely approximated to the median line. Owing to the great enlargement of these glands, and to the constant formation of a viscid mucous secretion which clogs up the throat, the respiration is often seriously interfered with.

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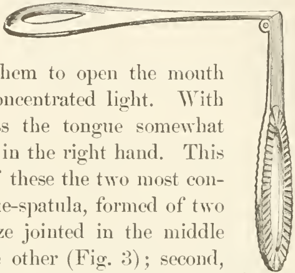
[1] Loc. cit., p. 52.

[2] Diseases of Children, p. 105.

[3] Dict. encycl., p. 20.

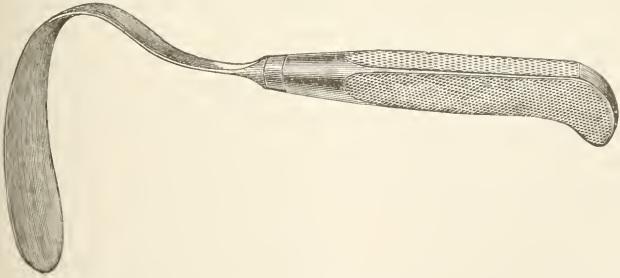
Fig. 3.



Folding tongue-spatula.

In order to examine the tonsils of most children, it is only necessary to get them to open the mouth widely, and to project into the throat a concentrated light. With many others it is also essential to depress the tongue somewhat forcibly by means of a tongue-spatula held in the right hand. This instrument presents numerous forms. Of these the two most convenient ones are, first, the articulated tongue-spatula, formed of two flat pieces of metal of slightly different size jointed in the middle and capable of being closed one upon the other (Fig. 3); second, the tongue-depressor of Sass (Fig. 4), in which there is a flat mouth-piece attached at nearly a right angle to a suitable handle. The mouth-piece is roughened or properly excavated, so as to hold the base of the tongue down more easily. When a child will not open its mouth, and resists obstinately all attempts at persuasion, it is necessary to hold its nares close together. At the moment it is then compelled to open its mouth in order to breathe, the tongue-depressor should be rapidly introduced between the teeth and carried backward to the base of the tongue, and this organ held down firmly until a thorough inspection of the tonsils has been accomplished.

Fig. 4.



Tongue-spatula (Bosworth.)

By reason of the propagation of inflammation to the cellular tissue around the lower jaw, and especially when it takes place near the articulation, the little patient can open its mouth only a short distance, so that at times it is quite difficult to make a direct inspection of the diseased parts. The effort of deglutition becomes extremely painful, and occasionally much viscid saliva dribbles constantly from the angles of the mouth. The pain of deglutition is sufficient at times to occasion spasmodic action of the muscles of the face and of those brought into action during the effort of swallowing. The soft palate is so much swollen that it cannot come into close contact with the posterior pharyngeal wall at this time, and the consequence is that the retro-nasal space is not completely separated from the middle pharynx, thus allowing a partial regurgitation of food or drink through the nasal passages. The voice becomes distinctly nasal, or is reduced to a mere whisper; the neck is moved with considerable difficulty, owing to the swollen condition of the deep parts; and the breath is intolerably fetid. Painful sensations in the ears are more apt to occur than in the preceding forms, and the danger of acute suppuration of these organs is greater. All the general symptoms of fever—headache, inappetence, aching pains in the limbs, restlessness, insomnia, etc.—are more pronounced than in the acute form previously described, where the inflammation has not involved the glandular tissue, at least to any great extent.

Course, Duration, Terminations, Complications, and Sequelæ.—In the great majority of children the disease never goes beyond the congestive stage, and generally terminates in resolution. In the mildest form it may last only twenty-four hours, and may scarcely excite more than a slight febrile movement. Such instances are likely to be those in which the tonsillitis is purely local and has been occasioned by some topical irritant. The mean duration of marked symptoms is from three to five days. At the same time that the general symptoms improve, the pain in the throat subsides, the deglutition becomes easier, and the tonsils are less inflamed and swollen. Usually at the end of a week or ten days all symptoms have disappeared and the tonsils have returned to their normal appearance and condition.

Whenever a tonsillitis goes on to suppuration, the glandular inflammation is accompanied by lancinating pains, and the formation of an abscess is preceded by well-marked, repeated rigors. It is very unusual for more than one tonsil to suppurate, and when both tonsils do suppurate they never do so at the same time, but the formation of abscess in the second tonsil takes place when the first one has become nearly well. Abscess of the tonsil usually points anteriorly towards the buccal cavity, and is likely to open spontaneously in this region. It may evacuate itself posteriorly, and has been known to show itself by a distinct swelling near the angle of the jaw, where it afterwards opened. Eustace Smith[1] declares that "if one tonsil only be affected, at the end of five or six days a yellowish spot can be detected on the reddened and glossy surface of the gland." It is at this point that the abscess subsequently bursts, at the expiration of a few hours or on the following day. In the rare instances in which one or both tonsils are said to have suppurated, so soon as the purulent collection opened the painful symptoms of the little patient were at once greatly relieved, and the cavity of the abscess healed afterwards very rapidly. In children the purulent contents of the abscess are usually swallowed, and not expectorated as in adults. According to Meigs and Pepper,[1] this statement is not invariably correct, since they speak of the sudden bursting of a tonsillar abscess after an effort at vomiting, or spontaneously, and of a gush of pus coming from the mouth.

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[1] Diseases of Children, p. 586.

It is extremely rare that a case of suppurative tonsillitis in childhood has, in consequence of any of its complications, terminated in death. Yet there are such instances on record. As an illustration I would cite the case of a girl,[2] thirteen years of age, who died of suffocation on the second day of the disease; and another, by Norton,[3] in which ulceration of the internal carotid artery caused an immediately fatal result in a little girl four years old. The complications and accidents accompanying and following suppurative tonsillitis in adults are relatively quite frequent. Such cases are reported by Montagne, Roche, Velpeau, Boisieri, Morgagni, Londe, Grisolle, Louis, Ehrmann, Lefort, Müller, and Way.[4] These cases, however, do not interest us at present, and I refer to them merely to establish the differences which separate parenchymatous tonsillitis in childhood from the analogous disease in adult life.

Paralysis of the soft palate and pharynx has been remarked many times after acute inflammatory diseases of the throat. Such cases among adults have been reported, notably by Maingault,[5] Gubler,[6] Alex. Mayer,[7] Germain-Sée, Tardieu, and Hervieux.[8] Among children the instances referred to are infrequent. Still, in an interesting article by Dr. Charles H. Knight[9] we find the following references: two interesting cases by Broadbent,[10] both in children, in one the larynx also being involved; another by Lichtwitz,[11] in a patient ten years of age, who rapidly improved under faradization; a third, reported by Dr. Knight himself, in a girl nine years of age, following a violent cold in the head: in this case "the tonsils and fauces were almost normal, and the oro-pharynx was congested to only a slight degree," while the nasal pharynx was intensely congested and swollen. Personally I have encountered such a condition in children on at least two different occasions, when the character of the acute tonsillitis was somewhat doubtful, but appeared to me of the nature of lacunar tonsillitis. When the paralysis of the soft palate manifests itself, the tonsillar inflammation has usually subsided. It is made evident mainly by two symptoms,—viz., the nasal intonation of the voice, and the difficulty of deglutition, with partial regurgitation of liquids or solids through the nasal passages. These two symptoms are due to the fact that the soft palate does not apply itself close to the pharynx during deglutition and during articulation of sounds which require this contact to be complete. The effort at deglutition may be more or less difficult. Usually when the alimentary bolus has passed the superior constrictor muscle it then passes downward without difficulty. The paralysis is said to affect especially that side of the palate which has been the seat of inflammation in connection with an attack of tonsillitis.

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[1] Diseases of Children, 7th ed., 1883, p. 364.

[2] Rilliet et Barthez, Traité des Maladies des Enfants, 1853, tome i. p. 227.

[3] The Throat and Larynx, London, 1875, p. 12.

[4] New York Med. Record, February 25, 1888.

[5] Thèse de Paris, 1853.

[6] Archives de Médecine, 1860–61.

[7] L'Union Médicale, Sept. 22, 1860.

[8] Bull. de la Soc. Méd. des Hôp. de Paris, t. iv. pp. 1858–1861.

[9] Paralysis of the Velum Palati in Acute Naso-Pharyngitis, New York Med. Jour., June 5, 1886.

[10] Trans. of the Clin. Soc., 1871, iv. 92.

[11] Revue mensuelle de Laryngologie, etc., Jan. and Feb., 1886.

There is a strong tendency in some children, after repeated attacks of acute tonsillitis, for the tonsil to pass into a condition of more or less marked chronic enlargement. In the development of this condition an hereditary predisposition may often be seen in different members of the same family.

Morbid Anatomy.—In acute inflammation of the tonsil the gland becomes swollen, owing to inflammatory exudation. In the superficial form this enlargement of the tonsil depends in a large degree upon the congestion and thickening of the mucous membrane. In lacunar tonsillitis there is a considerable increase of the secretions of the lacunæ, which results in considerable distention of these cavities and the formation of more or less concrete cheesy-looking masses. Frequently the mouths of the crypts are distended by prolongations of the large masses inside, and the aspect of the tonsil is then of itself characteristic. Occasionally the condition of the lacunæ is evident only when a section is made through the tonsil. These cheesy masses, besides the epithelial and pus cells and cells similar to those of the follicles, occasionally contain cholesterine crystals. As soon, also, as they become old, they are filled with numerous bacteria and micrococci, and give forth fetid gases, of which the butyric acid is best known. Owing to admixtures of different elements, the masses are not invariably white, but become more or less yellowish, or gray, or brown. In children I have never known them to become calcareous, but in adults this change is occasionally met with. J. Solis-Cohen[1] speaks of peritonsillar abscess frequently accompanying follicular tonsillitis, and of its being confounded at times with the suppuration accompanying the parenchymatous form. Such a combination I do not remember to have encountered in children, any more than the "distended follicles filled with whitish-yellow contents" mentioned by this author.

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[1] Pepper's System of Medicine, vol. ii. p. 385.

In parenchymatous tonsillitis the greater enlargement of the tonsil is due to the infiltration of the gland-tissue with the products of exudation which also affect the cellular tissue within and surrounding the tonsil. The adjacent parts are also markedly thickened by inflammatory deposits. The salivary glands and submaxillary lymphatic ganglia are often very sensitive and tumefied. The latter inflammation is more closely connected with that of the palate than with that of the tonsils, by reason of the more direct anatomical connection of their lymphatic vessels. The closed follicles are swollen, owing to increase of their contents, and their uniting membrane is softened. When suppuration is about to declare itself, these follicles burst their membrane and unite in the formation of small abscess-cavities, or a large abscess, implicating an extensive portion of the tonsil. In some of these instances of suppuration of the gland, pus has been found in the soft palate and between the muscles of the tongue at its base.[1] Inflammation and thickening of the coats of the internal jugular vein on the affected side have also been observed, together with the presence of pus and blood-clots in its interior, which offered a sufficient explanation of the swelling around the parotid and submaxillary glands. Repeated attacks of acute tonsillitis leave in many cases a condition of permanent hypertrophy of the gland, which is characterized by thickening and induration, mainly due to inflammatory hyperplasia of the submucous connective tissue. In those rare instances in which death has resulted from œdema of the glottis, the pathological changes in the ary-epiglottic folds peculiar to that disease have been found.

Diagnosis.—The diagnosis of the particular form of acute tonsillitis present is not difficult, as a rule, if we bear in mind the onset, the symptoms, and the appearances previously described. The cases in which error is liable to arise and to be of considerable importance are those of lacunar tonsillitis closely resembling true diphtheria. Usually they may be distinguished by the character of the deposit on the tonsils. In lacunar tonsillitis it is whiter, more elevated, and in small areas (at first situated over the mouths of the crypts). It is easily removed by a throat-probang or brush from the surface of the tonsil, and no ulceration of tissue is found beneath. In diphtheria the membrane is less white, more continuous, more adherent, and tougher. It leaves behind it, when removed from the tonsil, a raw, bleeding surface. These are the ordinary distinguishing features; but in some instances if the disease be seen only twenty-four or forty-eight hours after its onset, and the numerous small creamy deposits of lacunar tonsillitis have united to form a continuous membranous layer, or if this layer be tougher and more adherent to the tonsils than usual, as it may be in common membranous sore throat (?), then the difficulty of making a correct differential diagnosis may become very considerable. We may be obliged to remain in doubt as to the precise nature of the case during one or more days. As a fact of great practical importance, I have learned to believe that if in such instances the membrane be thoroughly removed from a moderate area of one or both tonsils, and if in twelve hours or less the membrane reform with its primitive characters as first observed by the attendant physician, it will surely indicate the tonsillitis of diphtheria. If the contrary be true, I am always encouraged as to the ultimate result in the case, and conclude that I have to do with lacunar tonsillitis, or a form of membranous sore throat which lacks, fortunately, the malignant features of true diphtheria.

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[1] Dictionnaire de Médecine et de Chirurgie, vol. ii. p. 128, Paris, 1865.

In determining the precise nature of the tonsillitis we should always carefully weigh the different constitutional symptoms. Yet there are two which will occasionally deceive us,—viz., the temperature and albuminuria. The temperature may be relatively low in very bad cases of diphtheria during the whole course of the disease, but we should not be deceived or put off our guard if the deposit in the throat have the characteristic signs. In diphtheria we usually expect to find some albumen in the urine; but here, again, we should not be misled by its absence if the local signs in the throat are of bad augury, since such exceptions will occur, as I have seen more than once. When, however, the membranous deposit in the throat is typical of diphtheria, when there is persistent elevation of temperature during several days, and marked albuminuria, there will be little doubt as to the malignant nature of the disease. With wholly different symptoms we can be equally confident of the innocent nature of the local affection, which will ordinarily turn out to be lacunar tonsillitis. For example, in follicular tonsillitis we may have a rapid and very high rise of temperature (104° to 105° F.), but this temperature will not last more than twenty-four hours. Albuminuria does not occur in this disease.

As to the membrane on the tonsil, whilst in the great majority of instances the distinctive characters which mark, on the one hand, diphtheria and, on the other, lacunar tonsillitis are sufficient to separate clearly the one from the other, yet now and then cases arise which baffle our closest study and observation. As Goodhart[1] remarks, however, most intelligently and, as I believe, correctly, of those cases in which membrane forms, "if one distinction may be singled out as less likely to mislead us in any disputed case of angina, it is to be elicited from the attentive observation of the behavior of the membranous formation about the tonsillar fauces."

There are cases, also, with severe constitutional symptoms, in which the tonsils are very red and swollen, without membranous deposit. Are they cases of non-contagious angina simplex, or of a sore throat which is the forerunner of scarlatina? Until the eruption of the latter disease appears, we are often in extreme doubt as to the proper diagnosis. Even when an eruption has appeared, it may be—on account of its irregularity in localization, duration, physical characters, or all combined—that we are still in legitimate doubt as to the nature of the disease with which we have to deal. Not long ago I saw a boy, about three years old, with fever, red, swollen tonsils, slight digestive disturbance, and a scarlatinous rash about the neck and chest. The boy had been taking no drug previously. These symptoms lasted for two days, and then disappeared under appropriate simple remedies. Since that time the boy has been perfectly well. In view of the absence of all sequelæ, I now consider this case one of moderate acute simple tonsillitis; but at the time I was uncertain as to whether the case was not one of mild scarlatina. In all such cases it seems to me the part of wisdom to express a certain degree of doubt as to the diagnosis, rather than to run the risk of ignoring wholly a grave disorder.

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[1] Diseases of Children, Amer. ed., p. 107.

From what precedes we may justly conclude that there are few acute diseases which demand more careful examination, more exact investigation, and greater exercise of good judgment than the sore throats of children. The criteria for the diagnosis of disease which are sufficient in our study are often quite inadequate in obscure cases which we frequently encounter in actual practice. Even to say, with Meigs and Pepper,[1] that "in some cases the diagnosis cannot be positively determined until the time at which the eruption of scarlatina makes its appearance has passed," is not altogether satisfactory, since during epidemics there may be real cases of scarlatinous angina[2] without at any time the characteristic eruption appearing, either on the face, neck, trunk, or limbs.

Again, the differential signs given by Eustace Smith are occasionally quite insufficient, as I have observed many times. This author states[3] that "the appearance of the inflamed mucous membrane is very different in the two diseases. In scarlatina it is more widely diffused, and of a more brilliant red, than at the beginning of quinsy; and on the soft palate the redness is usually punctiform, which is not the case in tonsillitis." Such distinctions, in obscure cases, in actual practice, will serve our purpose about as little as to say that in diphtheria the membrane is ash-colored and leathery or that there is early swelling of the cervical glands. These signs are then not present, or, if present, they are not accompanied by a sufficient number of characteristic signs, and of themselves are not pathognomonic; as the former may occur in other forms of membranous sore throat, and the latter I have seen in several instances of acute simple tonsillitis.

Lennox Browne makes a statement to which I have seen few if any exceptions, and which I regard as of great *practical* value in diagnosis, especially between diphtheria and lacunar tonsillitis: *i.e.*, the membrane in tonsillitis is limited to the tonsils themselves, whereas in diphtheria it is extremely rare not to see patches at the same time on the uvula and the soft palate.[4]

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[1] Diseases of Children, Philadelphia, 1883, p. 365.

[2] Trousseau, Clinique Médicale, Paris, 1877, p. 175.

[3] Diseases in Children, p. 588.

[4] The Throat and its Diseases, 2d ed., p. 230.

It may be useful to direct attention to the fact that occasionally tonsillitis has been confounded with laryngitis. This could occur only where the direct examination of the throat had been neglected, or the seat of the pain on deglutition and the vocal changes had been carelessly observed.

Prognosis.—As regards life, we may safely say that the prognosis of acute tonsillitis is almost invariably good. We need to allow only for those cases in which some extremely rare accident occurs, as in suppurative quinsy when hemorrhage or asphyxia has been the immediate cause of death,[1] and for cases where the fatal termination has revealed a grave error of diagnosis, as in cases called lacunar tonsillitis which were really diphtheria, or cases of scarlatinous angina which were said to be simple angina. The returns of the Registrar-General in England and the sanitary reports elsewhere would probably show numerous examples of these mistaken diagnoses.

Whilst acute tonsillitis may be, and usually is, entirely recovered from, there are numerous children of a debilitated and strumous constitution in whom permanent hypertrophy is apt to remain after one or more attacks. Again, in some children the susceptibility to recurrence of acute attacks of tonsillitis is very great and increases with every fresh attack. Especially after even slight enlargement of the tonsils has occurred, the advent of later attacks is caused by a trivial exposure or a slight digestive disturbance.

Treatment.—There can be little doubt that, as a rule, the first indication in the treatment of acute tonsillitis in children is to obtain a free evacuation of the bowels. This may best be brought about by one or two grains of calomel in tablet form, dissolved, or not, in a little water, and followed in three hours by a dessertspoonful of Rochelle salt in aerated Vichy. Small doses of sulphate of magnesium with quinine, repeated three or four times in twenty-four hours, are also very useful. The following is a good formula:

℞ Magnesii sulph., ʒ iii;
Quininæ sulph., gr. vi;
Acid. sulphurici dil., gtt. xx;
Syrupi zingiberis, ℥ ss;
Aquæ, ad ℥ iii.
M.

S. A dessertspoonful every three hours to a child three or four years of age.

It is almost always a good thing to keep the bowels slightly relaxed for a day or two. This statement is particularly applicable to those children whose bowels are apt to be constipated, and who rarely get a normal, healthy movement unless they are aided by some medicinal means. Whenever the tongue is coated and at the same time the bowels are not costive, an appeal

to the stomach directly by means of an emetic dose of ipecac in powder (from two to five grains for a young child) will often be followed by the happiest effects. It is true that this method of treatment is deemed somewhat harsh by over-anxious mothers: still, when it can be employed, I know of no better way of lessening active tonsillar congestion.

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When the bowels or stomach have been relieved in the manner recommended, resolution of the tonsillar inflammation will be accelerated by small, repeated doses of tincture of aconite root. From one-fourth to one-half drop in a teaspoonful of water, given at first every fifteen minutes, and later every half-hour or hour, will very soon diminish temperature and lower the pulse and respiration, while increasing the action of the skin, and thus promote speedy amelioration.

When the child is old enough to take them, benefit, both general and local, is obtained from guaiacum in the form of lozenges, or in mixture with a teaspoonful of glycerin. Cohen[1] advises the use of the ammoniated tincture of guaiacum topically, in the form of a gargle, with cinchona, honey, and chlorate of potassium. "The beneficial effects," he says, "will often be manifested within less than twelve hours."

Owing to the favorable action of guaiacum and of the salts of potassium and those of salicylic acid, Lennox Browne traces a strong analogy between quinsy and rheumatism. I have no doubt that the connection between these two diseases is very marked in some instances, but I am also confident that in very many cases no pathological connection of importance can be discovered. Still, if the salicylate or the benzoate of sodium be given early in the disease, it is often found to shorten in a remarkable manner the march of the inflammation and to prevent suppuration. By the use of the latter remedy the lacunar form of disease is said to be cured in from twelve to thirty-six hours, and without any local application. Boislinière reports twenty-five cases of this kind.[2] The efficiency of these remedies, especially in the formative period of the disease, is heightened when it is preceded by a full dose of an alkaline purgative. Oil of gaultheria may be used as a substitute for the salicylate or as an adjuvant to it.

During convalescence from quinsy a bitter infusion of cinchona, gentian, or quassia may be combined advantageously with an alkali, and will suit, as a rule, the sensitive stomach very well. Later a tonic of quinine and iron is more useful, in order to give strength and color to a little patient who is markedly anæmic. The following is a good formula:

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[2] Medical News, March 3, 1888, p. 237.

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When the bowels or stomach have been relieved in the manner recommended, resolution of the tonsillar inflammation will be accelerated by small, repeated doses of tincture of aconite root. From one-fourth to one-half drop in a teaspoonful of water, given at first every fifteen minutes, and later every half-hour or hour, will very soon diminish temperature and lower the pulse and respiration, while increasing the action of the skin, and thus promote speedy amelioration.

When the child is old enough to take them, benefit, both general and local, is obtained from guaiacum in the form of lozenges, or in mixture with a teaspoonful of glycerin. Cohen[1] advises the use of the ammoniated tincture of guaiacum topically, in the form of a gargle, with cinchona, honey, and chlorate of potassium. "The beneficial effects," he says, "will often be manifested within less than twelve hours."

Owing to the favorable action of guaiacum and of the salts of potassium and those of salicylic acid, Lennox Browne traces a strong analogy between quinsy and rheumatism. I have no doubt that the connection between these two diseases is very marked in some instances, but I am also confident that in very many cases no pathological connection of importance can be discovered. Still, if the salicylate or the benzoate of sodium be given early in the disease, it is often found to shorten in a remarkable manner the march of the inflammation and to prevent suppuration. By the use of the latter remedy the lacunar form of disease is said to be cured in from twelve to thirty-six hours, and without any local application. Boislinière reports twenty-five cases of this kind.[2] The efficiency of these remedies, especially in the formative period of the disease, is heightened when it is preceded by a full dose of an alkaline purgative. Oil of gaultheria may be used as a substitute for the salicylate or as an adjuvant to it.

During convalescence from quinsy a bitter infusion of cinchona, gentian, or quassia may be combined advantageously with an alkali, and will suit, as a rule, the sensitive stomach very well. Later a tonic of quinine and iron is more useful, in order to give strength and color to a little patient who is markedly anæmic. The following is a good formula:

℞ Quininæ bisulph., gr. xx;
Tinct. ferri chloridi, ʒi;
Syrupi zingiberis, ℥i·
Aquæ, ad ℥iii.
M.

S. A dessertspoonful before meals three times daily, through a glass tube.

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[1] Pepper's System of Medicine, vol. ii. p. 388.

[2] Medical News, March 3, 1888, p. 237.

among those who have healthier dwellings and more sanitary habits. The conditions to which I have just referred will often produce scrofula; and the frequent association of hypertrophy of the tonsils with this dyscrasia is readily explained when we consider the structure and functions of these glands. It is not infrequent, however, to discover enlarged tonsils accidentally and when neither previous disease nor objectionable environment will explain their existence. It is fair to assume with infants, when there exist simultaneously purulent ophthalmia, eczema, impetigo of the face and scalp, or nasal discharges, that these local diseases may have acted as efficient causes for the chronic enlargement of the tonsils.[1] I should be disposed ordinarily to believe that they were all local manifestations of the dyscrasia,—scrofulous or syphilitic,—though we could not say definitely that the size of the tonsils was much influenced by the presence of the other diseases as a matter of direct effect and cause.

Occasionally the child reaches puberty before tonsillar enlargement manifests itself, and it is then explained by some sympathetic connection between them and the sexual organs.[3] As regards sex, it is curious to observe the greater frequency of tonsillar enlargement in boys than in girls. Syphilis, hereditary or acquired, may cause enlargement of the tonsils, and this is also occasionally true, according to Guéneau de Mussy, of follicular disease of the pharynx. Children generally grow out of this condition of enlarged tonsils by absorption, or shrinking of the connective tissue, and at puberty it usually ceases to be a disease of much importance, although occasionally, as I have remarked before, the contrary is true. Even if the tonsils do not diminish in size at puberty, provided they remain stationary, they cease to cause annoyance to the individual, by reason of the increased dimensions of the throat and fauces as compared with their size during childhood. According to Bosworth, true hypertrophy of the tonsils probably never disappears except by excision, and has a far greater tendency to increase than to remain *in statu quo*.

Anatomical Appearances and Pathology.—The glands are enlarged and indurated. Both glands usually share this enlargement, but not always to an equal degree, as one gland is frequently larger than its fellow. They can be felt behind the angle of the jaw, and project into the throat on each side, between the pillars of the fauces. They have the appearance of round light-red tumors, of various size. Sometimes they are smooth and glistening on the surface, sometimes irregular and rough, from the openings of underlying distended lacunæ or ruptured follicles. They may be studded with a yellow, curdy secretion which exudes from the orifices of the crypts. They give more or less of a sensation of firmness and elasticity when pressed upon with the finger, which depends doubtless upon the degree of organization which the new fibro-connective tissue has reached (Sajous). They may be the size of a chestnut, of a bantam's egg, or even larger, and sometimes almost touch in the middle of the throat. According to Dr. G. V. Poore, the friction of the two bodies against each other may be a cause of superficial ulceration. In all cases in which the tonsils are notably enlarged, the adjacent mucous membrane is habitually congested and relaxed. Now and then we meet with cases in which one tonsil is enlarged and the other is of normal size. The tonsil may grow downward or upward, as well as inward. Adhesions between the tonsils and pillars of the fauces are not infrequent.[1]

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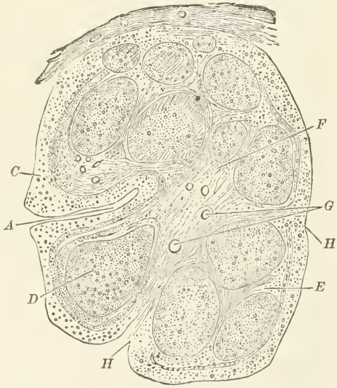
[1] Mackenzie, loc. cit., p. 60.

[2] Medical Times and Gazette, Sept., 1859.

The inter-follicular and deep fibro-cellular stroma is increased. Usually it is hardened, and resists section with the knife, giving out a creaking noise; occasionally it is soft and friable.

The walls of the crypts are thickened, and their cavities are dilated and filled with viscid mucus, or concretions of different degrees of consistence, usually caseous in children. The closed follicles are doubled in size, and generally increased in number. The entire mass of each tonsil weighs more than in the normal state; occasionally the growth has increased the weight by half an ounce or more (Chassaignac). Under the microscope the contents of the closed follicles are less transparent than in a normal state. Their epithelial cells are granular and increased in number. The morbid condition is a true hyperplasia, in which all the constituents of the gland are multiplied, thus causing the increase in size. The papillæ beneath the epithelial covering of the gland are often more numerous and less elevated than in the normal state (Mackenzie). The color of the cut tonsil is variable,—sometimes of a livid or dusky red, again of a sort of pale rose, brick-red, or yellowish hue. The capsule of the tonsil is thickened, indurated, or softened (E. Vidal), and the lymphatic ganglia of the neck (E. Owen) and under jaw are secondarily enlarged. The vessels of the connective tissue are enlarged; the acinous, mucous glands have disappeared.

FIG. 6.



SECTION OF THE ENLARGED TONSIL.—*A*, hilus; *C*, epithelial covering; *D*, lymphatic follicles; *E*, stroma: *F*, increased connective tissue of stroma; *G*, enlarged vessels; *H*, slight interruption of the epithelial covering.

According to Bosworth, there are two distinct varieties of enlarged tonsils: 1, the hypertrophic form, in which the glandular tissue is mainly affected and the tonsil is rough and irregular; and, 2, the hyperplastic form, in which there is increase solely of the fibro-cellular stroma and the tonsil is smooth and rounded. The first form is due to repeated attacks of catarrhal inflammation; the second belongs to the diathetic condition of struma, and is especially frequent in children. The second form tends to disappear at puberty, whereas the course of the first form is that of a continuous growth. Bosworth admits that these two forms are frequently combined in the same individual, but he also claims that it is important to recognize their independence of each other in many instances. Bosworth's clinical observations confirm the pathological researches of Dr. M. D. Mann.

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[1] It is important to bear this fact in mind whenever we contemplate excision of these glands. Prior to the operation the adhesions should be separated. We thus avoid wounding the pillars, which if cut will often bleed profusely.

Symptoms.—It is quite probable that many symptoms usually attributed to the presence of enlarged tonsils in children are independent of them. It is also true that when the tonsils are but slightly enlarged the symptoms of this condition are inconsiderable. Indeed, they are frequently limited to two,—*i.e.*, an increase of mucous secretion in the back of the mouth and a liability to take fresh colds. In infants enlarged tonsils have been known to make it almost impossible for them to retain hold of the nipple in nursing, and hence their proper nutrition was interfered with.

Still, there are unquestionably a few characteristic features which indicate their existence.[1] Among these we should first refer to the habit of loud snoring during sleep, which is a source of discomfort to themselves and of anxiety to their parents. Especially is this anxiety marked when, as is often the case, the child is very restless at night, and appears to be the victim of dreams which make it cry out, or talk in a very incoherent manner. This condition differs in no way from what is ordinarily termed nightmare, a phenomenon first described by Dr. Howard in 1873 in connection with tonsillar hypertrophy. The nightmare is a result of the obstruction of the faucial opening, which occasions imperfect respiration, hence the blood which supplies the brain is not sufficiently aerified, and the latter does not act normally. During waking hours, owing to increased muscular activity, this cerebral disturbance is not felt. A condition similar to the one referred to is met with in various diseases of the lungs, heart, and larynx, but in none of them do the attacks occur frequently in the same night, as they do in enlargement of the tonsils. Such children have a very thick tone of voice, which has also a nasal twang[2] that is very characteristic. The characteristic voice is due, no doubt, to several factors, among which the blocking up of the palato-pharyngeal opening and the interference with the movements of the tongue and soft palate are the principal. The snoring is explained by the narrowing of the fauces, which necessitates mouth-breathing in part, and thus occasions vibrations of the soft palate, particularly during inspiration. They are apt to be troubled with snuffles, and at times have a dry, hacking cough, which is very annoying, and is due to the habit of mouth-breathing, which renders the throat parched and irritable.

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[1] According to Dr. F. H. Hooper, of Boston, many symptoms formerly attributed to tonsillar hypertrophy may now be properly assigned to those indicative of adenoid hypertrophy in the naso-pharynx. (See Boston Med. and Surg. Jour., March 15, 1888.) I will write fully on this subject under the head of diagnosis.

[2] Bosworth says there is "absence of any nasal twang."

Deglutition is interfered with, owing to the diminished size of the faucial opening and to the fact that the action of the muscles which propel the food towards the gullet is rendered difficult. It is particularly uncomfortable on the recurrence of acute inflammation. Little children find it necessary to swallow only the most minutely divided portions of food. There is often a desire to take fluid very frequently during the meal, in order to facilitate the passage of the solid particles.

The senses of smell and taste are frequently impaired. Pain is a very rare symptom of tonsillar enlargement, even in adults, and I have thus far never met with a notable example of it in a child. Occasionally glandular enlargement in the neck may accompany chronic hypertrophy of the tonsils: in these cases it is more pronounced on one side than on the other. In advanced cases the breathing is constantly interfered with, and sometimes becomes labored upon very slight exertion. The interference with respiration, whilst it is mainly dependent upon the enlarged tonsils, is also increased by the swollen and relaxed condition of the adjacent mucous membrane.

The condition of the tonsils and of the surrounding soft tissues renders them peculiarly liable to the recurrence of catarrhal inflammation. Whenever one of these attacks occurs, it occasions increase of size of the tonsils, and also greater muffling of the voice and more intense dyspnœa. Often there is dulness of hearing, owing to thickening of the membrane lining the Eustachian tubes. Tinnitus aurium also occurs not infrequently, and occasions much distress. Formerly these aural symptoms were attributed to the pressure made by the enlarged glands upon the Eustachian orifices. This view, in my judgment, is not ordinarily correct, since the catarrhal thickening of the Eustachian orifice is frequently only the extension of an inflammatory condition of the naso-pharynx. In some rare cases it would seem as if impairment of hearing were due to pressure on the opening of the Eustachian tube by the enlarged tonsil. This statement is confirmed occasionally by the result of tonsillar excision, which carries with it evident improvement of the hearing and relief from tinnitus.[1] Even though these advantages may not proceed directly from ablation of the tonsils, we shall at all events have a clearer field for treatment of the part actually diseased. In examining the external auditory canal in these cases, we often find impacted cerumen.

Changes in the nose, the upper jaw, and the thorax are among the serious anatomical deformities caused by enlarged tonsils. The nose is pushed up by the palate, and thus the nasal passages become much obstructed and the organ itself has a pinched appearance. According to Semon,[1] the nostrils fail to be developed on account of want of use. In these cases the palate is relatively high and arched, the upper jaw does not acquire its ordinary dimensions, and thus the teeth are crowded and frequently overlap one another.[2]

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[1] Edmund Owen says "the hearing may not be improved immediately after the removal of the tonsillar mass." (See Surgical Diseases of Children, p. 192.)

As regards the slight development of the upper jaw and the pigeon-breast deformity of the chest, these are explained by a previous rickety cachexia rather than by any influence of the enlarged tonsils. It is, of course, true that when these organs are increased in size and obstruct the isthmus of the fauces they interfere considerably with a thorough expansion of the lungs. Still, it is difficult to believe that here is a sufficient explanation of pigeon-breasted children, and it appears more rational to admit the existence of rickets, which is at once the cause of the deformity of the jaw, of that of the chest,[3] and of enlarged tonsils. The deformity of the chest in these children was described first by Dupuytren in 1827,[4] and later by Shaw in 1841, who offered an ingenious theory to explain it. In his opinion, it was mainly due to the fact that the lungs were supplied insufficiently with air on account of the small faucial opening. The ribs were raised in each inspiratory effort, but not so much as was necessary, and consequently there was a tendency to a vacuum between the lungs and the chest-walls which finally resulted in a sinking in of the latter on either side.

The pressure of enlarged faucial tonsils has also been used to explain the existence of dilated anterior nares. According to Meigs and Pepper,[5] the sudden attacks of dyspnœa which afflict these children are evidence rather of some rachitic disease of the bones of the skull (craniotabes) than of enlarged tonsils. These suffocative attacks in children closely resemble those of laryngismus stridulus, and may occur without the coexistence of enlarged tonsils. The same is true of rickets itself, for enlarged tonsils are not an invariable symptom of this disease. Sometimes the attacks of dyspnœa are so intense that sudden fatal termination is feared. They may occur at meals, or at night during sleep. Such a case is reported by Wesley Mills in a child three years of age.[6]

The general appearance of children with enlarged tonsils is somewhat characteristic. They are usually pale, under-sized, and feeble in aspect. This condition is particularly noticeable when it is compared with that of other children in the same family who are exempt from this disease and who are often rosy and vigorous. The explanation of the meagre look of these children which is generally received is, that the enlarged tonsils interfere notably with the respiration and hence prevent in great degree the proper combustion of the waste products of the economy. In other words, the supply of oxygen is diminished, and, this element being one of the great excitants or stimulants of healthy nutrition, this latter fails proportionately to the lessened amount of the former taken up by the economy. This explanation is not altogether the correct one. What is more probable is that these children are peculiarly susceptible to chills (Eustace Smith), and that, owing to this fact, their stomachs are frequently the seat of a gastric catarrh which seriously interferes with the nutritive changes which are so essential to health. Certain it is that such children have a languishing air, with dark circles under the eyes. Their breath is foul; their movements are hard, light-colored, and extremely offensive. They are apt to suffer from considerable flatulence, with distention of the bowels. Infrequently there is a slight oozing of blood from the surface of the tonsils at night, which is afterwards found on the pillow. The posterior nares are much obstructed at times, so that nasal respiration on this account alone is extremely difficult. According to Lennox Browne (p. 236), the impeded nasal respiration causes the patient to snore loudly during sleep, and to breathe audibly when awake, with the mouth wide open. Difficulty of breathing, owing to the obstruction of the larynx, may occur in extreme cases, and thus produce collapse in the lower portions of the lungs. Under these circumstances the chest-walls undergo a characteristic deformity,—*i.e.*, the lower portion of the sternum recedes in a sort of cup-shaped cavity, while at the same time the upper portion of the sternum becomes very prominent. This change of the chest-walls should not be confounded with that due to rickets, where the chest falls in by reason of the softening of the ribs and the whole sternum becomes very prominent. In this change of configuration of the chest due to the enlarged tonsils, the lower portion of the sternum falls in on account of the yielding of the cartilages.

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[1] St. Thomas's Hospital Reports for 1883.

[2] Most if not all of these symptoms are now explained, according to Dr. Hooper, by the presence of adenoid vegetations in the naso-pharynx.

[3] Vidal claims to have seen the chest-deformity in children entirely exempt from rickets (loc. cit., p. 23).

[4] Repertoire d'Anatomie et de Physiologie

[5] Loc. cit., p. 367.

[6] Archives of Laryngology vol. iii. p. 62.

The fetor of the breath in cases of enlarged tonsils is often due to the offensive, decomposing cheesy masses which fill the crypts. Furthermore, a great deal of this decomposed material finally appears at the mouths of the follicles, especially when there have been successive acute exacerbations. In the acts of deglutition it is carried into the stomach with the food. In the passage of the inspired air over these fetid masses it becomes impregnated with unhealthy emanations. Inasmuch as these conditions last during long periods of time, it is easy to understand how the health will gradually fail, though vigorous in the beginning, and anæmia become established. According to Bosworth (p. 134), in these cases of enlarged tonsils the heart becomes affected with hypertrophous dilatation, as a result of impaired health and impoverished blood.

The cough which attends these conditions is often paroxysmal and distressing. Taken with the morbid appearance of the patient, it inspires one with anxiety as to the possible development of phthisis. This opinion is still further confirmed by an imperfect examination of the chest, since we may hear in the supra-spinous fossa (E. Smith) a somewhat hollow breathing sound, which, however, is in all probability transmitted from the pharynx. The idea of its being due to a consolidation of the lungs is immediately abandoned as soon as it is observed that this sound is not heard in inspiration, and that it disappears entirely when the mouth is open. There is, furthermore, no dulness on percussion over this same area. Lennox Browne[1] relates several instances in which a severe reflex spasmodic cough in children was due to enlarged tonsils. After numerous remedies had been vainly employed, the tonsils were removed, and then the cough disappeared.

Diagnosis.—This is usually determined by simple inspection of the pharynx. The tonsils may be seen to be augmented in size, and offer frequently a ragged or diseased appearance. Frequently they are enlarged, congested, and have a more or less regular globular appearance. The surface is then often smooth, if the degree of intercurrent catarrh be slight, and the orifices of the crypts are closed. If there be some acute accompanying inflammation, the lacunar opening may be plugged with epithelial products similar in nature to those found in follicular tonsillitis. In some children when the tongue is depressed there is more or less rotation of the tonsils, carrying their inner surface forward and making them appear larger than they really are. This appearance is heightened by the effort of retching, which is produced in a sensitive child even though the examination be conducted with great care. If the child be old enough to comprehend and carry out advice given, by urging it to inspire air forcibly we may be able to form a more correct appreciation of the exact amount of tonsillar enlargement present.

There are numerous cases in which the tonsils are in part concealed by the pillars of the fauces, and thus, although they may be considerably enlarged, mere inspection will not permit us to determine this important fact. We are then obliged, as Mackenzie advises, to recur to our tactile sensations in order to obtain accurate knowledge of the dimensions of these glands. To carry out this method of investigation, the index finger of the right hand should be introduced into the mouth and pressed directly against the tonsil, whilst that of the left hand makes external pressure behind the angle of the jaw. With the fingers of both hands in the relative positions described, we can readily form a correct opinion as to the absolute size of the tonsils. In all such cases, even before the foregoing examination has been made, a physician who is in the habit of examining throats, and who is therefore a good judge of normal appearances, will be willing to affirm that the tonsils are enlarged, simply from the knowledge which is afforded by direct ocular examination of the throat.

It is not very infrequent in children to have a tonsillar enlargement confounded with a retro-pharyngeal abscess. This disease may be differentiated by the fact that the swelling is situated on the median line, pushes forward the soft palate, and to digital examination is elastic and fluctuating. Occasionally the swelling of retro-pharyngeal abscess is diffuse, and shows no disposition to pointing. In these cases the differential diagnosis may be difficult, although the greater interference with deglutition, the choking attacks which occur, more marked dyspnœa, the return of fluids through the nose when attempts to swallow are made, and a more diffused swelling of the deep parts under the angle of the jaw, are signs of considerable value as indicating the pharyngeal disease. Since the publication of Dr. Hooper's able article,[1] I have directed more attention to the differentiation in children of difficulty of breathing formerly ascribed by me to tonsillar hypertrophy. In cases in which doubt exists, the diagnosis of adenoid vegetations may be made either with the mirror, or, what is usually far preferable, by means of the index finger of the left hand passed into the naso-pharynx, whilst the right hand supports the head of the child. The presence of the abnormal growths can be surely discovered by this method of examination. As to their precise influence in causing dyspnœa and the other symptoms described as belonging to tonsillar enlargement, that can only be determined in any case by an operation by which these growths shall be removed. If we can rely wholly upon Dr. Hooper's experience, it would appear as if former views held in regard to the pernicious effects of tonsillar hypertrophy were frequently erroneous, and that these results were the direct outcome of adenoid vegetations. In a discussion of Dr. Hooper's paper, which was read by the author before the New York Academy of Medicine, I held the opinion, which I have had no reason to change since, that adenoid vegetations are *relatively* uncommon in New York City, and are not in this place responsible, as a rule, for the symptoms described by Dr. Hooper as occurring very frequently in Boston and elsewhere and as being occasioned by these growths.

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[1] Loc. cit., p. 236.

Prognosis.—So far as life is concerned, the prognosis is not serious. Indeed, at times in children enlargement of the tonsils, apart from the fact that it renders them more liable to contract colds, cannot be regarded as a grave affection.[2] When, however, we estimate the increased gravity which attaches itself to all acute diseases of the respiratory organs in children who are affected with tonsillar enlargement, we must make a graver prognosis. In many instances, as we have shown, the increased size of the glands is the evident cause of numerous diseases which manifest no tendency to disappear unless the tonsils be removed. Besides, in those cases in which operative measures, for one reason or another, cannot be employed, the tonsils remain without decrease of size during many years, and are but little influenced by ordinary therapeutic measures internally or locally. It is true that if left alone these glands will usually decrease in size towards the age of puberty. Previous to that period, however, they occasion so much annoyance and interfere so markedly with processes of nutrition that suitable means should be adopted for their early reduction or complete removal. According to Bosworth, no treatment except complete removal or complete destruction is of any avail against the true hypertrophic tonsil. This author admits, however, that in the case of a hyperplastic growth, if we see it at an early stage, we may hope by judicious medication to promote absorption of the already effused material. Even in these cases, if the enlarged tonsil be the evident cause of impaired nutrition by reason of interference with sufficient aeration of the blood, with the digestive function, or with quiet sleep, the operative procedure becomes imperative. Meigs and Pepper speak of cases in which treatment is successful in reducing the enlargement. In our experience these cases are usually most obstinate, and often require prolonged local and constitutional treatment to obtain even very slight favorable results.

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[1] Loc. cit.

[2] In early life, Mackenzie regards the disease as one which almost always requires immediate attention.

Treatment.—The general or constitutional treatment of chronic tonsillar enlargement is of importance not so much in reducing the size of these glands as in promoting healthy nutrition in the child. Usually children thus affected suffer from symptoms of gastric catarrh. Their tongue is ordinarily coated with a white or yellowish fur, the bowels are torpid, and the slightest exposure to cold, or any undue fatigue through study or late hours, is apt to cause an exacerbation of the stomachal derangement and likewise an attack of acute catarrh affecting the tonsils.

The first counsels to such children should be to wear a broad flannel band around their stomach during the day, to bathe daily in cold water, to wear thick-soled shoes, and to be very careful in their diet and also in regard to the regular daily movement from the bowels. All sweets and excess of farinaceous food should be avoided. Broths, milk, eggs, roast and broiled butcher's meat, stale bread, should form the principal articles of their diet. Whenever there is an acute gastric attack, no remedy is so effective as an emetic of ipecac. Afterwards small doses of tincture of ignatia restore the tone of this organ. If the bowels are sluggish, they should be moved with compound liquorice powder, or emulsion of castor oil, or some other equally useful aperient. As the child improves and as soon as his stomach appears in sufficiently good condition to bear them, cod-liver oil and iron, quinine, and other such remedies should be given, so as to tone up the system and increase its powers of active nutrition. At times a small quantity of good claret or port wine at meals will aid digestion and increase flesh and bodily vigor.

Lennox Browne attributes some importance to the use internally of sulphide of calcium and iodoform in reducing the size of the tonsils. Half a grain each, repeated several times a day, is the proper dose for a child. This action appears to me doubtful. For my part, I have never known any internal remedy have much manifest effect in lessening the size of these organs, despite the fact that some tonic remedies unquestionably affect favorably the growth and nutrition of the child.

Whenever there are marked evidences of struma or of rickets, of course our choice of the proper internal remedy will be influenced by the opinion we hold in regard to the treatment of either of these diseases. The iodide of iron is a specially useful tonic in such cases, as is also the lacto-phosphate of lime. Unfortunately, this preparation, like Parrish's chemical food, contains so much sugar as occasionally to upset the stomachs of the children who take it, by its undergoing acid fermentation owing to the excess of gastric secretions which are present in the stomach. Whenever the chronic enlargement of the tonsils is accompanied by evidences of lacunar inflammation, as shown by the whitish, cheesy deposits at the mouths of the follicles, the underlying diathetic condition should be appropriately treated by means of guaiacum or chlorate of potash.

When the health of the child is good, and particularly if the tonsillar hypertrophy be of recent date and moderate in amount, and if the gland has a relatively soft consistence, remedies such as chloride of ammonium, chloride of calcium, and iodide of potassium may be employed in small and repeated doses (combined or not with the vegetable alteratives like phytolacca, stillingia, and sarsaparilla), and are occasionally useful in reducing these glandular enlargements (Cohen). Lambeau, when other means had failed to produce good results, has seen the greatest improvement result from the use of sulphur-water internally and in local douches.[1]

Of course, whenever it is possible, such children will thrive better in pure country air, or near the sea, than in the vitiated atmosphere of a city or large town. Showering the neck daily with cold water seems to render the child less susceptible to the recurrence of attacks of acute angina.

Local Treatment.—Occasionally the application *externally*, behind and below the angle of the jaw, of tincture of iodine or of the compound iodine ointment has apparently been of some service in promoting the resorption of these enlarged glands. Of more obvious service, however, are the applications of modifying agents directly to the glands themselves. Among these we should mention particularly the daily application of tincture of iodine.[2] Equal parts of tincture of iodine and liquor potassæ (E. Smith); powdered alum; glycerite of tannin; powdered alum and tannin, equal parts, applied with the insufflator (Sajous), or, better still, with a moistened pharyngeal spatula (M. Mackenzie), Fig. 7; nitrate of silver in solution (ten or twenty grains to the fluidounce), or the lunar caustic in stick, have all been recommended.[1] Personally, I have found it useful to paint the enlarged tonsils two or three times a day with tincture of chloride of iron and glycerin, one or two drachms to the fluidounce. This application is continued during several weeks. Again, where children are somewhat older and will permit it, the use of nitrate of silver in thin stick (Gordon Holmes), or fused on the extremity of a roughened aluminium probe and inserted in the lacunæ of the tonsils, is of unquestionable service. These applications are scarcely painful, and may be repeated every three to six days, depending upon the degree of local reaction. The objection to nitrate of silver is the continued unpleasant taste it leaves behind in the mouth during several hours. The applications of iron are objectionable on account of blackening the teeth, and, where there are gold fillings, of doing positive injury to the teeth themselves.

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[1] Rapport de M. Blache à l'Académie de Médecine, Avril, 1861.

[2] The spasms of the glottis caused by these applications, spoken of by Seiler (see Diseases of the Throat, 2d ed., p. 189), I have never seen. Bosworth properly directs attention to the fact that compound tincture of iodine is freely soluble in water, whereas the ordinary tincture is not: hence we shall obtain good results in producing absorption of tissue from the former diluted with water that we cannot expect from the latter diluted with water (loc. cit., p. 135).

Fig. 7.

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Pharyngeal spatula (Mackenzie).

I have no experience in the intra-parenchymatous injections of solutions of dilute acetic acid (M. Mackenzie), carbolic acid (Cohen), or iodine and ergotin, by means of a hypodermic syringe, which have been recommended by some writers. It may be that in certain rare cases they will be found useful. If these injections be employed, the point of the syringe should be thoroughly embedded in the substance of the tonsil, and from three to five drops of fluid injected. The iodine solution may or may not be further diluted with water; the ergotin may be of the strength of one drachm to the ounce.

These injections may be repeated about once a week, or according to the effect produced. They are somewhat painful in certain subjects, and should be introduced carefully and gently. Frequently the pain of these injections is such, and their repetition is so dreaded by the child, that we are obliged to abandon them. Moreover, Eustace Smith[2] declares that he has "never seen a case where the glands have been appreciably diminished by this means." On the other hand, Seiler[2] states, "Injections of solutions of iodine into the substance of the gland by means of a hypodermic syringe are often followed by a speedy reduction of the gland, without causing the unpleasant results that are apt to follow the application of the drug to the mucous membrane." From this opinion I feel obliged to express my dissent.

Under the above methods of treatment I have occasionally known the enlarged glands to diminish in size; but this is not the rule, for too often the best directed, most persistent general and local treatment will remain unavailing. Under these circumstances, if the symptoms occasioned by the presence of the tonsillar enlargement be of such a kind as strongly to indicate its utility, excision of the hypertrophied portion of these glands should be unhesitatingly resorted to. The symptoms which, in our opinion, are sufficient to justify the operation are notable interference with normal hearing, defective speech, frequent irritative cough, general malnutrition and an impoverishment of blood, deformity of the face and chest-walls, dyspnœa, spasmodic attacks of choking at night, or persistent insomnia and restlessness. Of course we must always bear in mind the fact that many cases of enlargement of the tonsils are favorably modified in time, and especially towards the age of puberty. This consideration, however, will have weight only when the child has slight local or general disturbance, and when the age makes it necessary to wait but a few months, or a year or two at most, to see the result of the changes thus spontaneously brought about. We allude to it more because of the fact that some parents are strongly opposed to any operative interference.

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[1] Sajous believes that nitrate-of-silver solution acts rather to promote the growth of the tonsil by direct stimulation than to diminish its size. (See Diseases of the Throat and Nose, p. 289.)

[2] Loc. cit., p. 590.

[3] Loc. cit., p. 189.

Before leaving this question of local treatment, I would refer to two methods of treatment which, when we are able to carry them out effectively in children, are quite as useful as when adopted (as they ordinarily should be) for adults. These consist (1) in the use of the galvano-cautery and (2) in cauterization with chromic acid.

With a convenient handle, suitable points, and a reliable battery, moderate tonsillar enlargement may be reduced sufficiently to be considered normal. The applications are, as a rule, very slightly painful, and even this slight pain may be reduced to none at all by the local application to the tonsils of a four-per-cent. solution of cocaine with a throat-brush. There is no necessity to use a mouth-gag, if the operator is skilful and the child obedient. Under contrary conditions the operation had better not be attempted, as it must result in failure.

If the tonsil be scarified in two or three places with the cautery, the useful result of these transcurrent cauterizations can be increased by the application, on these burned surfaces, of a saturated solution of chromic acid, applied by means of a flattened or round metallic probe roughened at its extremity. A pointed glass rod may also be used for the same purpose. The chromic acid should not be employed too liberally, and any excess of it should be removed before an effort of deglutition takes place. Further, an alkaline spray, like that of Dobell, may be projected against the cauterized parts, as an additional precautional measure against possible swallowing or absorption of chromic acid, which would be objectionable on account of its poisonous qualities.[1] I am of opinion that chromic acid is the best of all chemical caustic agents hitherto employed in the treatment of enlarged tonsils, but, like all very active agents, it asks for care and delicacy in its use. Dr. Donaldson, of Baltimore, is in the habit of making small incisions into the tonsils and inserting afterwards a crystal of chromic acid into each cut.[1] The galvano-cautery, followed or not by chromic acid, has been successfully used by Drs. H. H. Curtis, Charles H. Knight, etc.[2] In a few instances of soft enlargement of the tonsils, Cohen[3] speaks of the possibility of reducing their size by means of electrolysis. He confesses, however, that this method is tedious and that "in some instances the results have not been worth the trouble of the performance." I doubt if one child in hundreds would permit the continued introduction of needles into the tonsils, by which alone this method can be carried out. As compared with the London paste,[4] which has been somewhat extensively employed by Dr. Morell Mackenzie and others in the removal of enlarged tonsils, I regard chromic acid as a far more efficient agent. I have found, with some observers, that the treatment of enlarged tonsils by London paste, although sometimes effective, is both painful and tedious.

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[1] According to Bosworth, the risk from absorption of chromic acid is *nil*, by reason of its instant combination with the albuminoid elements of the tissues and its reduction to the form of an inert and insoluble oxide of chromium. (See N. Y. Med. Jour., March 10, 1888.)

As to the good effects of massage of the tonsils, referred to by Starr in the American edition of Goodhart's treatise on Diseases of Children,[5] I can but be extremely sceptical. None less than a phenomenal child could be expected "to do this himself" and repeat it "for three or four minutes several times a day."

Operative Treatment.—This consists in the removal of the tonsils, entirely or in part, by the knife, tonsillotome, cold-wire écraseur, or galvano-cautery loop. One of the foregoing methods is doubtless in a certain proportion of cases rendered absolutely imperative. When the operation has been performed, the immediate or near result is most gratifying and remarkable. The child breathes better immediately; his appetite, power of deglutition, sleep, and general nutrition rapidly improve. The congested condition of the pharynx, which previously was a source of much discomfort and inconvenience, quickly disappears. The clearness of speech and the brightness and gayety of the child are often markedly increased. The operation may usually be performed rapidly, without much difficulty, with little or no physical pain to the child, and with little or no danger in the vast majority of cases. Except some difficulty in swallowing (due to soreness of the raw surfaces, and lasting usually only a few days) or possible secondary hemorrhage, there are no accidents to be apprehended.

There may be a few instances in which the bistoury and forceps are preferable to the guillotine for the excision of tonsils, even in children. Such, for example, are cases in which the tonsillar enlargement is small, irregular, and not easily grasped with the ring of the guillotine, or, again, in which the operator feels that he will be able more certainly to remove just the amount of tonsil that he thinks is required. In the great majority of instances, however, the tonsillotome is preferable to the knife, by reason of the ease and safety with which it can be employed.

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[1] Cohen, loc. cit., p. 233.

[2] N. Y. Med. Jour., Sept. 24, 1887.

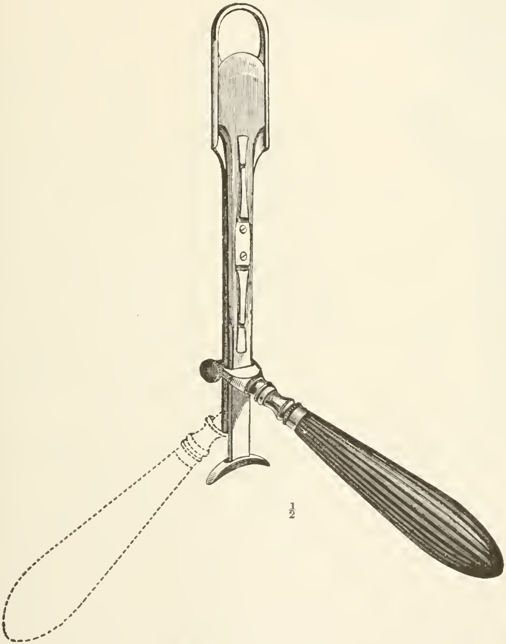
[3] Loc. cit., p. 233.

[4] London paste is composed of equal parts of caustic lime and soda with alcohol.

[5] P. 110.

There are very many kinds of tonsillotomes. Those most commonly in use, however, and which are most recommendable are slight modifications of Physick's or Fahnestock's tonsillotomes. The former, which is now generally known as Mackenzie's tonsillotome, and which has been somewhat modified by this physician, is probably the safest and most efficient instrument we have. It is strong, easily manipulated, and rarely fails, in the hands of a tolerably skilful operator, to cut away a suitable portion of the tonsil. This tonsillotome is here represented (Fig. 8). It consists essentially of a flat piece of metal with an elliptical opening at the distal extremity and a broad semicircular blade which when pushed forward closes the opening and cuts off the tonsil. In addition there is a stout handle at its lower part, which greatly facilitates the application of the instrument and enables the operator to hold it firmly against the side of the throat. Mackenzie has modified Physick's original tonsillotome so that the handle can be applied to either side of the shank of the instrument. This arrangement enables the operator to use the instrument on either side of the throat,

Fig. 8.

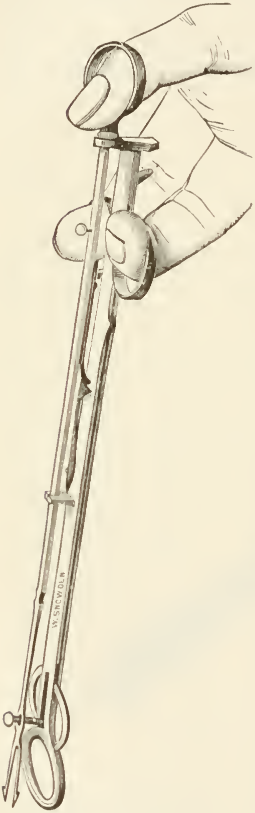


Mackenzie's tonsillotome.

and the free surface of the blade in each case is directed towards the centre of the mouth.

I also show an instrument after the model of Fahnestock, which, modified as it is at present, is usually known as Mathieu's tonsillotome (Fig. 9). This instrument is complicated, readily gets out of order, is kept clean with difficulty, and, although when it works well it is used with one hand and in a very rapid and satisfactory manner, may not cut through the tonsil, and in this case, from the way the pronged forks are made, has to be cut or torn from the tonsil. This somewhat troublesome and annoying occurrence took place once when I was operating with a new and apparently serviceable instrument.

FIG. 9.

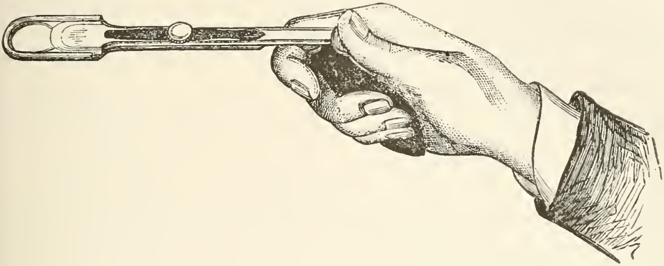


Mathieu's tonsillotome.

In order to make use of Mackenzie's tonsillotome, it is almost essential to have an assistant, who will steady the child's head, placing one hand on each side. The child should be in a straight-backed chair, with the face directed towards the light. Frequently it is essential to use some kind of simple mouth-gag to keep the child's mouth open while the tonsil is being engaged in the tonsillotome. One of the simplest gags, and also one of the most useful, consists of a round piece of wood ten to twelve inches in length and about the diameter of the middle finger of an adult hand. This may be held by the assistant between the posterior molar teeth and the side opposite to the one on which the operation is to be performed. As a rule, to prevent the enlarged tonsil from slipping out of the ring of the guillotine, it is necessary for the assistant to exercise slight pressure just beneath and behind the angle of the jaw, with the extremities of two or three fingers. The operator, seated directly opposite the child, then introduces carefully the tonsillotome so as not to include any adjacent parts in the ring of the instrument, and passes the elliptical opening over the tonsil to be excised. Holding the hilt of the instrument firmly with the right hand and in such a manner as to press the distal portion to the side of the throat, the blade, which was previously drawn back, is rapidly pushed forward with the thumb of the right hand and the tonsil cut off (Fig. 10).

It is quite sufficient, ordinarily, that a portion of the tonsil be excised. The rule adopted by careful practitioners is to take away that part of the enlarged tonsil which stands out beyond the pillars of the fauces. To extirpate more than this amount is unnecessary, and undoubtedly exposes the patient and operator, at times, to profuse and even dangerous hemorrhage. Some physicians dislike to use Mackenzie's guillotine, for the reason that they are afraid lest the portion of the tonsil which has been excised should fall into the throat and occasion symptoms of strangulation. There need be no such fear, especially if a slight twist or rotary movement is given to the distal extremity of the tonsillotome after excision of the gland, as this effort will surely carry the tonsil forward into the mouth, whence it can be spat or taken out.

FIG. 10.



Tonsil guillotine, in position for operating on the left tonsil (Browne).

It is unwise, in most cases, to give an anæsthetic for the operation of tonsillotomy if the patient can be managed without it. It complicates matters considerably, and makes quite slow and troublesome an operation which without it is rapid and simple. If the bleeding is to be so severe as to cause the operator anxiety, an anæsthetic prevents his having the active help of the child which is so valuable in avoiding the running down of blood into the larynx and trachea. The further objections to the use of chloroform and ether are such, in fact, as hold good in any other operation in which they are not positively necessary. As to the use of nitrous oxide gas, I would say that, while it is not followed by nausea or prostration, its effects are too transient to allow of this operation being properly performed in all cases. In some instances, at least, the patient wakes to consciousness before the work is finished. Of the different anæsthetics which may be employed, I consider ether the best, ordinarily. I should give to it my preference under most circumstances, rather than to either chloroform or nitrous oxide. The chief reason for using any anæsthetic is that we may avoid the struggling of the child at the sight of the instruments and from fear of the operation. The pain is usually very slight, and even if at times it be quite severe it lasts but a very short time. Whenever it seems desirable, this pain may be prevented almost entirely by the local use of cocaine.

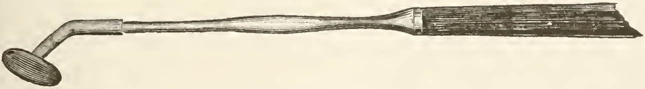
The fear of troublesome hemorrhage is unquestionably one of the leading reasons, if not the most important one, why tonsillotomy is so frequently postponed or not performed at all, and that, too, when the operation is plainly indicated by the sufferings of the child, and after the means previously employed for the relief of the morbid condition have proved to be insufficient. Now, this impression in regard to the risk of hemorrhage is either a legitimate and wholesome fear, or simply the dread which occasionally takes hold of timid practitioners in regard to the performance of certain operations, however free from real danger. I take the latter view. It is probable that few if any well-authenticated cases of death following tonsillotomy *in children* can be found in medical literature. On the other hand, it is true that grave hemorrhages, or at least hemorrhages sufficient in amount to cause much anxiety, have been not very infrequent. Hemorrhages the amount and duration of which have been inconsiderable, but which have caused the operator to feel nervous and worried, have been quite common. Of course, viewed coolly and deliberately from a statistical point of view, no one, it would appear, should feel very much dread in face of such a showing. Despite this statement, however, my observations would go to show that many practitioners dread to perform tonsillotomy and desire to pass the operation to some expert in throat-diseases. This feeling is not so evident in regard to other operations, some of which are generally considered to be of a more serious nature than tonsillotomy. I believe one explanation of this fact is because tonsillotomy has usually been talked about as if it were a trivial operation and without danger to the patient. This opinion as to its seriousness being propagated by physicians and received by parents, there results a corresponding action on the part of the latter. When the father or mother brings a child to consult the specialist, and the latter discovers a pair of large tonsils which should unquestionably be removed, he feels that in amputating them he is doing a thing which may possibly involve him *instantly* in considerable trouble and anxiety, and the outcome of which always seems to him a little uncertain. This contingency is apt to influence him, even though he may not wish to appear to regard it too closely, lest he get the reputation (a most unsavory one) of being an unnecessary and foolish alarmist.

When hemorrhage takes place after tonsillotomy, the rule is that it begins immediately, lasts a few moments only, though quite abundant during this time, and then stops either spontaneously or by giving the child a few mouthfuls of cold water, or by its sucking ice continuously for several moments. Sometimes these means are ineffectual, and the blood continues to flow from one or other tonsil quite rapidly. It is better, as a rule, therefore, to use some strong styptic preparation as soon as the tonsil is removed. The most effective of these is probably the one now known as the tanno-gallic acid gargle of the Throat Hospital Pharmacopœia. The formula for this gargle is as follows: Acid. tannic., gr. cccIx; acid. gallic., gr. cxx; aquæ, ℥i. Half a teaspoonful of this fluid must be slowly sipped, according to Mackenzie, at short intervals. It will almost always control the hemorrhage very soon. In some few cases the tendency to bleed will show itself repeatedly during the first few days after excision of the tonsils. In one case Lennox Browne[1] has seen the saturated solution of tannin fail to arrest tonsillar hemorrhage, and in this instance he substituted with success the "styptic colloid," a combination of collodion, alcohol, and tannin. This preparation applied directly to the bleeding points had the desired effect by causing a firm coagulum. Even in such exceptional cases as the foregoing the hemorrhage can almost invariably be checked by recourse to the tanno-gallic mixture.

Despite this statement, I consider it prudent to give here, especially for the benefit of the young and inexperienced practitioner, a few points of judicious counsel, which, if followed, will prevent his getting into any really serious trouble after a performance of this operation. 1. Never, when in conversation with the parents, make too light of the possibility of hemorrhage following tonsillotomy. 2. Never excise a tonsil without having a competent assistant to render timely service if required. 3. Always have ready near by (in addition to the tanno-gallic gargle and some pieces of broken ice) one or more pairs of long artery-clamps, a few sponge-holders, and a thermo-cautery.

A more useful instrument, however, in my judgment, than any of these, is a long metallic holder, with a convex metal button somewhat larger than a penny projecting from its distal extremity, supported by a firm metal rod half an inch in length (Fig. 11). Around this button a thick layer of sheet spunk may be wrapped or tied tightly. Armed with this compressor, one can feel tolerably safe after the excision of tonsils, since, even if the bleeding persists and we are unable to seize and twist the bleeding point (if a small artery be wounded), after removing the clots from the back of the throat, if necessary, we can at least exercise efficient pressure inside of the mouth. This instrument is far preferable to different kinds of double clamp pressure forceps which have been described. As to its superiority over pressure with the finger or the holding of a bit of ice against the bleeding surface, there can be no question with any one who has ever attempted to keep a finger in place in the back of the throat of a bleeding, struggling, and thoroughly frightened child. And if any one has once

FIG. 11.



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[1] Loc. cit., p. 240.

experienced the feelings which are uppermost in one's breast under like circumstances, he will never afterwards be disposed to ridicule the fears of one who writes about the unpleasant features or possibilities of the operation. Of course, if the hemorrhage from the tonsil be venous or capillary, instead of being arterial, the utility of a pressure instrument is even more unquestionable than ever. If further aid be required, it is simple enough to supplement internal pressure by counter-pressure made with the fingers on the outside of the throat.

An excellent measure for dealing with troublesome hemorrhage has been published by Dr. R. J. Levis, of Philadelphia.[1] An ordinary tenaculum is passed firmly through the tissues at the base of the tonsil, and the instrument is then given a decided twist. The torsion effectually compresses the oozing vessels, and is maintained by closing the patient's jaws on the handle of the tenaculum as it projects from the mouth. The jaws are then bandaged securely together. As a *dernier ressort* the actual cautery may be employed, or the carotid may be tied; but I doubt if these means will ever be required when prudence has been exercised as regards the size and kind of tonsil one removes with the tonsillotome.

It is always more prudent in excising tonsils not to remove the second tonsil until all hemorrhage has ceased from the surface of the one originally extirpated. With this precaution, it seems proper, as a rule, to remove both tonsils at a single sitting rather than to oblige the child to return for a second operation on another day.

It is worthy of remark that, in many instances where the tonsillar hemorrhage is difficult of arrest, it appears to be either venous or capillary in character. This sort of hemorrhage is often kept up by a tight collar or cravat, which constricts the neck and interferes notably with the venous circulation of the face, neck, and head. Again, some obstruction in the nose or naso-pharynx, such as thickening of the turbinated bodies, a deviated or swollen septum, or mucous polypi, may seriously interfere with respiration, and in this way tend to prolong unduly the hemorrhage. The practical deductions to be made from these facts are: 1, loosen all tight garments about the neck or chest; 2, let the patient open his mouth and breathe freely through it if the nasal passages are obstructed from any cause.

It should also be mentioned, since it has several times occurred in adults, that in cases where all means employed have failed to arrest tonsillar hemorrhage, an attack of syncope has obviously been the most important factor in saving life.

A means for arresting tonsillar hemorrhage which has proved so successful in the nose and womb may be tried if other means fail,—*i.e.*, the use of the hot douche by means of a Davidson's syringe. To be of any utility, the douche must be as hot as can be borne, and it must be intermitted occasionally so as to note the effect produced by its employment.

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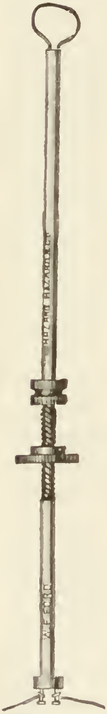
[1] Medical News, December 8, 1888.

Whenever the tonsils in children are of unusual size, whenever they are very hard or dense in structure, and when in addition the child is very weak, pale, and emaciated, or has already shown signs of a hæmophilic tendency, it is wise, in case tonsillotomy is considered necessary, to use either the galvano-caustic or the cold steel wire. By employing either of these methods all risk of profuse hemorrhage is surely avoided. Under these circumstances it would be advisable to administer an anæsthetic. Ether is preferable, because safer than chloroform, and because its anæsthesia lasts longer than is possible with nitrous oxide gas, especially as the latter has to be withheld during the operation. But if the incandescent wire be employed and ether be used for anæsthesia, great care must be taken to avoid ignition of its inflammable vapor. As soon as the child is completely under the influence of the ether, the bottle containing it and the ether-cone should be carried to some distance from the patient's head. The snare is then properly adjusted around the tonsil, a suitable gag is introduced, and the current is passed into the wire. Traction on the heated wire should be made only while the current is being passed, and this should be done in an intermittent manner, so that the tonsil shall not be burned through too rapidly and thus bring about the very accident we wish to avoid,—*i.e.*, hemorrhage. The platinum wire should be heated to only a dull-red heat, as otherwise (*i.e.*, if heated to white heat) it is liable to break, or to cut through the tonsil too quickly. In applying the platinum wire around the tonsil, care should be taken not to include too large an amount of the tonsil, as without this precaution we run a risk of cauterizing deep tissues, and when the slough comes away some days after the tonsil has been removed, possible secondary hemorrhage may result, or injury to surrounding important and healthy parts might occur. The objections to the use of the galvano-caustic snare are: 1, the necessity of being provided with a suitable electrical apparatus; 2, despite great care on the part of the operator to see in advance that everything is in proper working order, there is a possibility that at the critical moment the current may give out, or the wire break or become twisted.

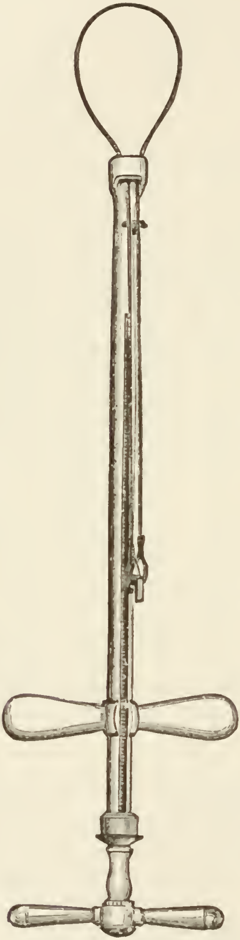
In case the cold steel wire is employed (and to this we give the preference, as a rule), no special precautions are called for in the administration of ether to the patient, except such as are required in all operations in the throat. Still, the snare has its drawbacks. 1. It requires some little time to cut through the tonsil. 2. There is considerable dragging oftentimes upon the surrounding soft tissues. 3. I have known the wire to cut partly through the tonsil and then remain embedded in the tonsillar tissue, until finally the operator was forced to cut off the portion of the tonsil inside the encircling wire with a pair of scissors and afterwards pull off the tightened snare. Unless the écraseur itself is quite stout and resisting, the shank of the instrument is liable to bend into the form of a letter S under the powerful traction exerted. If the cold-wire écraseur be employed, that known as Jarvis's écraseur is most to be recommended. It should be provided with a sufficiently powerful milled nut, and should be armed with No. 3, 4, or 5 piano-wire (Fig. 12). In some instances I can well believe that the wire-loop écraseur depicted by Browne (Fig. 13) will answer admirably for the same purpose, "and quite obviate all risk of hemorrhage."

Fig. 12.



Jarvis's snare.

Fig. 13.



Wire-loop écraseur for enlarged tonsils (half measurements), after Browne.

When a large tonsil has been removed by one of the instruments referred to, and after the patient has recovered from the effects of the anæsthetic (if one is used), the subsequent guidance of the patient is a relatively simple matter. For several days he should remain in the house, in order to avoid cold or atmospheric changes, which might bring on inflammatory changes of the tonsillar region or of the pharynx. An emollient gargle, or demulcent lozenges, may frequently be used to diminish faucial irritability or soreness and thus promote the comfort of the little patient. The diet should be mild, non-stimulating, and essentially composed of those substances, liquid or solid, which the patient can swallow with ease. If after the operation the portion of the tonsil which has been left in the mouth becomes covered with a membranous layer, which is occasionally the case, or shows slight tendency to heal, this condition will be favorably modified by occasional applications of nitrate of silver, iodine, or tannin. In order to avoid the recurrence of inflammation in the glandular mass which remains behind, it is essential that the little patient be watched somewhat carefully. Daily cool bathing followed by friction of the entire body, and attention to keeping the feet dry, to keeping away from cold draughts of air, and to simple diet, are very important.

The tendency to fresh outbreaks of tonsillar inflammation after removal of the hypertrophied portion has led some physicians to question the utility of the operation. While I do not believe such an argument should have much weight with reflective minds, I recognize that this sequence, unless carefully guarded against, may occasionally occur and bring a valuable measure into disrepute. In some children, despite all the care that can be exercised, tonsillar inflammation will recur after excision. In just such cases we are disposed to admit the important influence of an underlying rheumatic dyscrasia.

ADENOID GROWTHS OF THE VAULT OF THE PHARYNX.

By HARRISON ALLEN, M.D.

While the lymph-bodies of the vault of the pharynx are not separated by any sharply-defined line from those seen elsewhere in the respiratory passages, it is customary for clinical study to treat of them under a distinct head. These bodies, when hypertrophied, impede nasal respiration, excite inflammation, and maintain an increased flow of mucus from adjacent surfaces.

In addition to lymph-bodies, neoplasms, which are papillomatous in nature, arise from the same general region. The present essay deals chiefly with the structures last named. The clinical conditions arising from their presence are similar to those arising from the presence of the foregoing bodies.

Czermak,[1] while making examinations for the detection of the causes of difficulty in the use of the Eustachian catheter, made the observation that the instrument at times was deflected by reason of a number of adventitious masses in the naso-pharynx. He appears to have recognized a comb-like form which was attached to the posterior wall of the space. Türck and Semeleder subsequently confirmed Czermak's observation. Voltolini[2] recognized the growths and essayed their removal in the course of a treatment for deafness. B. Löwenberg[3] described the clinical bearings of the masses in connection with diseases of the ear. The first precise account of the condition was written by W. Meyer.[4] Excellent descriptions have appeared since that date in various text-books and in numerous clinical essays.

Histology.—The growths as seen in this country are true papillomata with an extensive lymphoid parenchyma. The lymphoid base is the same as "cytonene," or "adenoid," and is found in the lymphatic glands as well as in the Malpighian corpuscles of the spleen.

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[1] Der Kehlkopfspiegel, etc., Leipzig, 1860.

[2] Allgem. Wien. Med. Zeitung, No. 33, 1865, 265.

[3] Archiv für Ohrenheilkunde, 1867, ii. 103; also Gazette des Hôpitaux, Paris, 1878, li. 474, sq.

[4] Hopitals-Tidende, Nov. 4 and 11, 1868, 177. A translation of this paper appeared in Med.-Chir. Trans., London, 1870, 191.

Description.—The naked-eye appearances of adenoid growths are those of masses which, while, as a rule, pediculated and varying in size from that of an almond-kernel to that of a grain of wheat, may be nearly sessile—*i.e.*, may have a base greater than any diameter—and be uniformly rounded and small.

Adenoid growths are of a reddish color, of fleshy consistence, and very vascular. After removal they lose their color, and appear as hard, gray, seed-like bodies. The mucous membrane appears to be free at the base of each mass, and forms its pedicle. Pendent, as a rule, from the vault, on a line with the fossa of the Eustachian tube, the growths may lie posterior to the fossa,—namely, in the depression known as the fossa of Rosenmüller, or upon the parts which are parallel to the posterior wall of the pharynx. The writer has never seen them on the alæ of the vomer or on the borders of the posterior nares, though, according to some authorities, they may here occur. The growths appear to spring, in the main, from the mucous membrane covering the localities where connective tissue fills in the inequalities of the base of the skull, especially between the basilar process of the occipital bone and the ossa petrosa of the temporal bones, as well as along the line of the synchondrosis between the occipital and sphenoid bones. The variety of the masses usually coming under observation is the pediculated. In late childhood and early adult life the broad-based growths alone are seen.

While it is true that sessile vegetations may be met with, nevertheless many which appear to be sessile when seen in the rhinal mirror are really pediculated, as demonstrated by digital examination.

The pharyngeal bursa can occasionally be demonstrated lying in the midst of a number of adenoid masses, by a probe (which is passed through the nose) being received into its open mouth.

As above stated, the vegetations may persist until early adult life. G. W. Major[1] lays stress upon this fact. I have seen them retained as late as the twenty-sixth year. Others mention their being seen at the forty-fifth year. The masses thus seen are not large, but, while not creating obstruction in breathing, may excite and maintain a catarrhal state of the membranes both of the nose and of the pharynx.

Etiology.—Adenoid growths are papillomata which contain normal lymphoid tissue of the vault of the pharynx.

The neoplasm may be congenital, when it is difficult to escape the conclusion that it has been in some way associated with the canal which is found in early fœtal life penetrating the brain-case and uniting the anterior part of the pituitary body to the lining membrane of the pharynx.[2]

The writer has removed a congenital growth of the size of a filbert in a child six weeks old. The naso-pharynx was completely obstructed, and the difficulties attending the act of sucking early attracted attention to the condition. As a rule, however, the growths do not invite scrutiny in infancy, and, from the fact that most cases come under notice after the fifth year, it is probable that the papillomata at the vault are apt to take on a hyperplastic condition at or a little later than this time. The permanent teeth are beginning to erupt, and the altered proportion of the face and associated parts announces the change from infancy to childhood. The vascularity of the roof of the pharynx is increased, and should an attack of diphtheria, scarlet fever, or measles supervene, the growth may create the characteristic symptoms. That it may do so in the absence of any such complication producing cell-proliferations is, of course, tenable.

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[1] Medical Record, 1884, 569.

[2] Landzerk.

The fact that the growths originate before the synchondrosis between the basilar process of the occipital bone and the body of the sphenoid bone has closed renders it probable that some connection exists between the two conditions. In a number of crania which the writer has examined with reference to this subject, he has noted, in instances of irregularity of union at the suture, exceptional arrangements of the venous canals which pass from the pharynx to the nose. But, in the absence of clinical histories of the specimens, the statement is suggestive only. In a gentleman of twenty-five who came recently under the writer's care for an irritative form of pharyngeal and nasal catarrh he found that the angle or "fault" between the occipital and sphenoid bones was very deep, and the depression was completely filled with the adenoid growths.

Moderate hyperplasia of the lymphoid tissue at the bases of the papillomata of the vault does not in itself induce distress. The writer has detected the growth in children who never complained, nor had any symptoms been discerned by the parents. Out of twenty-one healthy Indian girls of the Lincoln Institute of Philadelphia examined by him, seven exhibited the growth. In four of these children it was small, in two of moderate size, and in one as large as a chestnut. No complaint was made in any of these subjects. Dr. W. Franklin Chappell[1] has recently examined two thousand children in the schools of New York, and found the growth in sixty. Meyer had previously examined one thousand school-children in Copenhagen, and found the growth in one per cent.

It is probable that the shape of the vault may determine whether or not the growth, normal in other respects, interferes with the functions of the pharynx. When the angle formed between the basilar process and the sphenoid and vomerine structures is of high degree and the axis-tubercle prominent, a mechanical disadvantage exists for the naso-pharynx to maintain its patency; on the other hand, when an angle of low degree between the parts named is associated, as is apt to be the case, with an inconspicuous axis-tubercle, a growth which is readily discerned may excite no symptoms.

Social, sexual,[2] and climatic influences appear to be of little importance.

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[1] American Journal of the Medical Sciences, 1889, 148.

[2] Writers generally agree with this statement. Yet Dr. Chappell (*loc. cit.*) found, in sixty examples of the growth, forty-nine in boys and but eleven in girls.

The growths are frequently hereditary, since the pre-existent states on which they depend are transmissible. Frequently all the children of a family will exhibit the growths and at least one parent the favorable shape of vault.

Adenoid growths are often associated with hypertrophy of lymphoid tissue elsewhere in the respiratory tract, especially in the oro-pharynx at the tonsils and the base of the tongue. As the tonsils are frequently enlarged at the time the growths of the vault are threatening the health, a common cause, in a measure, must be assigned for both, inasmuch as both hypertrophies have a physiological limitation and gradually recede in bulk with the maturity of the system. This remark does not apply to the basilingual lymphoid nodules, which are most pronounced in the adult and may persist throughout life.

Connection between adenoid vegetations of childhood and the etiology of nasal catarrh of the adult is difficult to prove. Yet, when an adult gives a history of mouth-breathing and tumescent tonsils which includes the period of early childhood, when together with this history the hard palate is found acutely arched, the epiglottis laterally compressed, and the existing nasal catarrh is of the congestive type, no reasonable doubt can be entertained that the catarrh is a survival of the juvenile disease and is caused by a neglected adenoid growth.

Signs and Symptoms.—The symptoms of adenoid growth can be placed under five heads,—namely, respiration, secretion, speech, hearing, and general condition.

The respiration-effects are direct and indirect. The direct effects are seen in the obstruction of nasal and the establishment of oral respiration. This does not occur unless the growths occlude the nares, the naso-pharynx, or the posterior naso-pharyngeal aperture,—that is to say, the opening between the velum and the posterior wall of the pharynx. When the mouth-breathing has been of long standing, the superior dental arch ordinarily is contracted and the roof of the mouth elevated. In some cases the patient breathes through the nose during the day, and the mouth is open only during sleep. Or the nasal respiration is normal in summer, and at all times when the patient is free from catarrh. During a sea-voyage the patient may be entirely comfortable.

The writer has frequently noted the way in which the mouth was narrowed and elevated in adults in whom no history either of adenoid disease or of nasal obstruction was given. In some of these persons unusually severe attacks of scarlet fever and diphtheria have been reported. It is probable that in such cases the vascular structures of the superior maxilla have been permanently changed during the protracted angina and stomatitis attending these diseases. Dr. Chappell (*loc. cit.*) believes that the exanthemata often inflame and enlarge adenoid growths which otherwise would have proved innocuous. The shape of the chest is distinctive of prolonged interference with normal respiration. Anteriorly the ribs are prominent, the sternum is angulated forward at the manubrio-gladiolar junction, and grooved at the gladiolo-xyphoid junction. A saucer-shaped depression is often found at the lower costal cartilages. The lower angle of the scapula projects. While the ribs are separated far from each other anteriorly, they are so closely pressed together posteriorly, especially at the lower part of the chest, as to have the intercostal spaces practically obliterated. Owing to the exceedingly narrow proportions of the upper part of the chest in the region of the shoulder-joint, the head of the humerus and the coracoid process are exceptionally prominent, and the clavicle is more than usually sigmoid in form. The antero-posterior diameters of the thorax are lessened. The curves of the sides are exaggerated. The open hand of the observer can clasp the side of the chest. Upon percussion the range of hepatic dulness is diminished on the chest-wall, but increased in the epigastric region; the sounds of the heart are modified, the first sound being shortened and the second lengthened, so that the two sounds appear to be of equal volume.

Sleep is disturbed. The patient snores, and wakens frequently to moisten the parched mouth and lips. Decubitus varies. It may be normal, or the body may rest prone, with the forehead resting on the flexed arms. In very young children the head often lies over the arm of the nurse. In sucklings the act of taking the breast is characteristic, since the mouth, being occupied by the nipple, can be but momentarily closed. Immediately after seizing the nipple the lips are withdrawn and a deep inspiration is taken, followed by a fretful cry.

The indirect respiratory effects are seen in a disposition to laryngeal stridor or cough. A croupy inhalation often follows an attempt at digital examination of the naso-pharynx. Sometimes the slightest touch of the region, or even a few drops of liquid thrown into the space, will be followed by a croupy cough. Occasionally asthma and æstival attacks of the so-called "hay-fever" coexist with the other symptoms.

Excess of secretion of mucus in the pharynx is always present. Tenacious mucus or muco-pus adheres to the walls of the naso-pharynx, to be occasionally dislodged. Young children swallow this material, and it is not often seen. In older subjects it is raised by hawking and is ejected. The enforced rest of the nasal chambers does not favor a normal condition of their lining membranes. The nostrils are ordinarily occupied with inspissated mucus. Occasionally, however, especially in children of a strumous habit, a true nasal catarrh is established. The pharynx is irritable. Use can be made of the act of gagging in determining the character of the mucus in the naso-pharynx, for at the moment of elevation of the velum and adduction of the palato-pharyngeal folds a thick drop of grayish mucus is seen protruding back of the uvula.

One of the more serious complications which may arise in the course of the distresses connected with adenoid disease is due to the inflammation of the masses in diphtheria, scarlet fever, and typhoid fever. In illustration the following case may be cited. A boy, aged thirteen years, who came under the writer's care through the family physician, Dr. Wharton Sinkler, exhibited in a marked degree the symptoms of adenoid growth. In addition to mental apathy and sullenness, the child had never been subjected to wholesome discipline. As a result he resisted treatment, which after a time was suspended. A few months afterwards the child sickened with typhoid fever. All the symptoms of adenoid growth were exaggerated during the illness; the mental perverseness increased and added greatly to the gravity of the situation. The child died on the fourteenth day. Dr. Sinkler was of the opinion that the unrelieved condition which accompanied the pharyngeal state undoubtedly contributed to the fatal issue.

Interferences with hearing are often met with. They arise from pressure of the growths against the orifice of the Eustachian tube, or its obstruction with mucus. Inflammation of the lining membrane of the middle ear not infrequently occurs. The impairment of the hearing will oftentimes awake the anxieties of the parents, although the other symptoms of the presence of the growths have long antedated it. Mutism may be due to adenoid growths, since the deafness arising from their presence may be absolute. Impediments of speech are present in proportion to the obstruction of the naso-pharynx, and are due to mechanical conditions. The resonance of the voice is diminished; the quality of the sounds dependent upon an open pharynx (*i.e.*, the naso-pharynx and the oro-pharynx acting as one chamber) is destroyed, and others are substituted which normally belong to a closed pharynx (*i.e.*, the naso-pharynx being separated from the oro-pharynx by the velum). Thus, *m* is changed to *b*, and *n* to *d*. The *l* and *r* sounds are muffled, since both demand a patulous condition of the upper air-spaces.

Adenoid growths appear to have little effect in maintaining stammering, if the writer can form a conclusion from two cases of stammerers whose condition in this particular was not improved by removal of the tumors.

The general state of the patient is affected in many ways by the presence of the growths. The imperfect rest and the unnatural breathing create anæmia, which, occurring in the system at an age when the growth-forces are actively engaged, produces a variety of disturbances, chiefly in the direction of functional disorders of the thoracic and abdominal viscera and of the nervous system. The patient has a capricious appetite; enuresis is occasionally noted; palpitation of the heart may be a prominent symptom. Chorea, especially of the facial muscles, is not unfrequently present. The disposition is often sullen or fretful, the memory is bad, and for these reasons (apart from the impairment of hearing) the child is often found to be a dull scholar. Drowsiness during the day may be complained of, though this may ensue upon interrupted sleep. In one instance, where in an adolescent the adenoid masses were associated with enlargement of the tonsils and engorgement of the cervical lymphatics, the writer assumed that the pressure of the enormous swellings against the carotid arteries might be held answerable for the symptom last named. Headache is often complained of in students. The symptom is of low grade and is limited to the forehead and temple. The effect upon the general development is noteworthy. In young children dentition is retarded. In adolescents puberty is delayed, as instanced in the retention of the juvenile voice. It is almost needless to add that attempts at vocal training are ineffective so long as the resonating chambers are closed.

The lymphatic glands of the neck are often swollen, but not in any significant way. They may suddenly enlarge without apparent cause. After they have thus changed, all the symptoms of adenoid growths are exaggerated.

Diagnosis.—The diagnosis of adenoid growths of the vault can be easily made by the aid of the rhinal mirror, or by the insertion of the finger into the naso-pharyngeal space. It frequently happens that the irritability of the pharynx is so great, or the discipline of the patient so poor, that the digital examination is the one which is alone available. The methods of conducting such an examination are as follows. In a young child the entire trunk, including the arms, should be wrapped in a broad towel or folded sheet. The child is held by the thighs, and is turned upon its back towards the lap of the physician in such wise that the head is held between the knees of the last-named while the lower part of the trunk is on the lap of the attendant. Thus the face is directed upward and the mouth can be easily opened by a jaw-dilator. The physician can hold this instrument with the left hand while inserting the index finger of the right hand into the naso-pharynx. If the growths are present he can feel them distinctly. The contrast between the slightly elevated, firm, obscurely plicated surface of the normal lymphoid tissue and the movable, pediculated, velvety grape-like masses of the papillomata is conclusive. When the finger is withdrawn it is found stained with venous blood, and the lower pharynx is also covered with blood of the same character.

Precaution is essential to demonstrating that the hypertrophies are the causes of the distresses reported,—namely, to eliminate all possible obstruction in the nose, the naso-pharynx, and the oro-pharynx. Obstruction in the nose, as illustrated by atresia, undue narrowing, etc., can be detected by inspection. Occlusion of the naso-pharynx by other than adenoid growths can be determined by the differences in the sensations given to the finger by the post-velal examination. Fibroma springs from the sides of the pharynx rather than from the vault, and is apt to involve the nares. It is always broad-based. From carcinoma and sarcoma the growths are distinguished by absence of local pain and of spontaneous hemorrhage, as well as by the differences in palpation. The above-named conditions are not apt to occur at the age at which adenoid growths are frequent.

Until the researches of Meyer the symptoms of adenoid growth were accredited to the tonsils. These curious bodies have been accused of many things of which they have proved themselves to be quite innocent. Hypertrophied tonsils may aggravate the symptoms of adenoid disease, but they never create them.

A falling backward of the tongue in sleep in the weakened state of the system following diphtheria and whooping-cough may simulate the mouth-breathing and snoring of adenoid disease. Such symptoms can be eliminated by careful examination of the nose and the naso-pharynx, conjoined with inquiry into the clinical history.

It is of interest to distinguish between the oral conditions due to the habit of thumb-sucking and those incident to faulty breathing. In mouth-breathers the incisor teeth of the upper jaw are vertically disposed or nearly so, instead of being inclined slightly forward and downward as is the rule in health. The central incisors are often inclined a little towards each other, and may even overlap. In thumb-suckers the incisors are very obliquely disposed from behind forward and from above downward, and protrude somewhat beneath the upper lip. The dental arch is always flat.

Prognosis.—The future of a case of adenoid growths when neglected is somewhat as follows. The child after passing the fifteenth or sixteenth year begins to breathe through the nose, the tonsils gradually lessen in size, and the disposition to nasal catarrh and earache disappears, though a sufficient degree of vascular excitement may persist in both nose and ear. The reflex symptoms no longer annoy. The shape of the chest, the elevated roof of the mouth, and the contracted dental arch remain unchanged throughout life. As already remarked, the future is often gravely complicated by illness, and doubtless the presence of adenoid growths may determine a fatal issue in scarlet fever, diphtheria, etc.

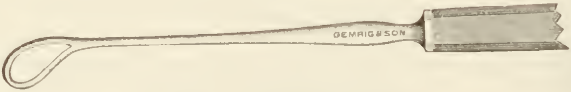
Under treatment the prospects are very favorable. When the growths are removed, the condition of the child changes for the better in a few days in a manner that is very striking. The face becomes animated, the appetite improves, and the reflex symptoms disappear. The growths never return.

Treatment.—It is evident from what has been said that a small adenoid growth may exist in the naso-pharynx without exciting distress. The only danger which may arise from the neglect of such a growth is the remote one of an increase of severity of an angina accompanying scarlet fever or diphtheria. On the other hand, if the group of signs and symptoms recognized as associated with adenoid is present, then the method to be pursued is to secure as prompt a destruction of the masses as is practicable.

If the patient is an infant it should be etherized and the growth ablated. This is best done by the finger, inserted as in digital examination of the naso-pharynx. In older children a choice of treatment is presented. The growths can be removed under ether by ablation with the finger, or rasped away by curettes introduced through the nose, or picked away by forceps inserted through the nose or the pharynx; or they may be absorbed by local applications of drugs, or destroyed by caustics. Advocates of each of these procedures have written upon the subject. In place of entering into a discussion of these various plans, the writer will assume the responsibility of stating that the drift of opinion is decidedly in favor of removal of the growths by the finger, curette, or forceps, rather than securing their destruction by absorbents, astringents, or caustics. Assuming, therefore, that the two last-named procedures may be ignored, the question to answer is, which of the ablation-plans of treatment is the best? Capart, Dalby, and F. H. Hooper use the finger,—either depending upon the finger-nail to scrape away the growth, or strengthening the nail with a shield as recommended by the two authorities first named. The writer accepts the method of operating with the unguarded finger as the best. The sense of touch is of great use, and it is withheld by any other method. It is efficient, and no criticism can be brought against it. The statement sometimes made, that the growths are sometimes too firm to be broken down by the finger, is not sustained by the writer's experience. The liability for masses detached by the finger to fall into the larynx appears to be an exceedingly remote one.

The details of the manipulation are as follows. The child is etherized. The jaws are separated and fixed by a gag. The body is brought to the sitting position, with the head a little flexed to induce the blood to flow forward out of the mouth and the nose. The person etherizing can readily manage these details, if a third person, acting as attendant, assists. The operator passes the index finger through the naso-pharyngeal aperture (*i.e.*, back of the velum), and, turning the palmar aspect of the tip downward, extends the terminal joint, and by such a manipulation squeezes the growths successively between the finger and the firm bony vault from which they spring. If one or more growths are so movable as to prevent this manipulation being easily accomplished, the edge of the vomer can be used as a resistant surface against which the growths can be crushed. The contents are forced out of their limiting membrane, and the pedicles shrink back against the vault and, as a rule, can be ignored. Sometimes a pair of polypus-forceps, or others of special design recommended by Löwenberg and J. Solis-Cohen, can be used to seize growths which lie on the posterior wall of the pharynx near the velum or the sides of the naso-pharynx. (Fig. 1.) When the main growths are removed, the curette or ring-knife recommended by Meyer and modified by Beverley Robinson can be inserted through the nose, and, by employing the index finger of the right hand as a guide to the surfaces of the naso-pharynx which it is desired to rasp, all remaining tags of the masses be thus removed. In the writer's hands instrumental aids are secondary to the use of the finger, and they can often be dispensed with.

Fig. 1.



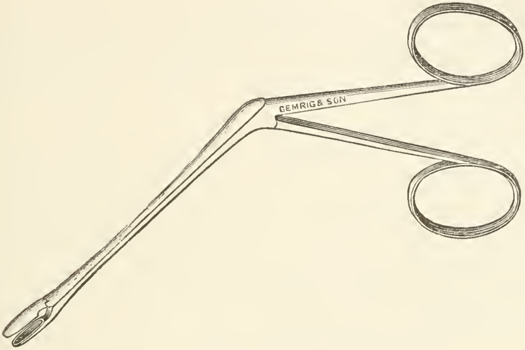
Curette for removing adenoid growths.

Störck uses a guillotine-snare, which must certainly leave a vascular irritable surface beneath it unless each growth be reached above its pedicle.

Without the aid of the sense of touch this is difficult to do. If the instrument requires the insertion of the finger for its adjustment, the snare is not needed. The same remark applies to the use of the galvano-cautery snare.

The administration of ether is sometimes impracticable, owing to heart-affection, or is for some other reason held to be undesirable. Under these conditions the use of forceps is necessitated,—either Farnham's modification of the old alligator forceps (Fig. 2), used through the nose, or the post-nasal Löwenberg-Cohen forceps, inserted back of the velum. But either of these methods is less accurate and more tedious than the immediate ablation under ether.

FIG. 2.



Farnham's forceps for removing adenoid growths.

It is undoubtedly the case that the longer the neglect the more painful become the growths. In several young adults who came under the writer's care, attempts to remove the masses without ether were followed by evidences of acute suffering. As a rule, considerable hemorrhage follows the ablation. The blood flows forward, for the most part, and always ceases spontaneously. But often enough is swallowed to excite nausea and vomiting a short time after the operation. The contents of the stomach, stained with the blood which has become darkened by contact with the gastric secretions, often alarm the attendants, but this emesis is entirely salutary, and the patient directly thereafter becomes composed. Acute frontal headache and sometimes a reflex toothache ensue after operation, but both symptoms soon disappear.

The child should be carefully housed, and for three days kept in bed. This is often a difficult matter, for by the following day the patient is so comfortable that the chief care of the nurse is to carry out these instructions. In a word, no feverish reaction is to be anticipated. The diet for a few days should be fluid or semi-solid, to protect the teeth and jaws from the labor of mastication.

Cases of secondary hemorrhage have been observed, but they are rare. The writer has never seen one. If instruments are inserted through the nose, a secondary epistaxis may more frequently occur: in the few cases observed by the writer the bleeding stopped spontaneously.

In young children the results of treatment are secured at once. But in adolescents the long habit of faulty articulation and of mouth-breathing will oftentimes persist. Such children are improved by a course of calisthenics, and by being placed under a good trainer of the voice in speech. If the mouth remains open during sleep, a leather chin-piece can be adjusted to straps passing round the head, to keep the jaws in contact.

STENOSIS OF THE LARYNX.

By CHARLES E. SAJOUS, M.D.

If the conditions included under the above heading taken in its literal sense were alone to be treated in this article, it would be necessary to limit ourselves to disorders producing constriction or narrowing (στενός, "to make narrow," "to contract"), and omit the consideration of foreign bodies of the larynx, which do not cause obstruction by contracting the glottis proper, but by offering a mechanical impediment, varying with their size and form, to the free passage of air. We would thus be deprived of an important element in the discussion of the general subject, and defeat our purpose of making this paper as complete as the limited space at our disposal will permit. The subject of obstruction of the larynx by foreign bodies has therefore been introduced and treated as freely as though properly comprised within the limits of the title of this paper.

When we consider the larynx anatomically and physiologically we can but conclude that Nature, though so fertile in her efforts to protect the organ against the intrusion of foreign substances during deglutition, was less successful in devising means by which the effects of disease in limiting its all-important function in relation to respiration could be compensated. Indeed, we find ourselves furnished with duplicate organs of sense; with pairs of many of the viscera performing important functions,—the lungs, in relation to respiration; the kidneys, in relation to micturition; the ovaries and testicles, in relation to reproduction, etc.; while the larynx is not only single, but has a double function to perform,—respiration and voice-production, the latter in itself standing as an etiological element of danger in the production of conditions calculated to compromise its integrity.

Topographically considered, from epiglottis to cricoid, the internal aspect of the larynx presents features which would seem to render the presence of local disorders much more serious, as regards the maintenance of life, than in other situations. Principal among these is the narrowness of the passage, which makes it possible for an inflammatory disorder, practically benign in other localities, to jeopardize life by even a moderate infiltration; while a tumor, of a size that would hardly cause anxiety when located in other parts, would here compromise the possessor's existence to a material degree. Again, muscular spasm might involve, as in general chorea, every superficial muscle of the body without danger to the sufferer; in the larynx spasm becomes, on account of its limited lumen, a frequent cause of sudden death. Foreign bodies, owing to the antagonistic action of the inspired air-current against the physiological closure of the epiglottis, readily gain access to the laryngeal cavity, to become embedded above or below the ventricular bands, which soon swell, further securing the intruder against extraction and increasing the danger of suffocation to which the patient is exposed.

Histologically, the richness in cellular tissue which characterizes the upper part of the larynx renders it liable to dangerous infiltration upon provocations which elsewhere would prove unimportant; while pathologically, its situation between the lungs and the upper air-tract causes it to take part to a greater or less extent in the inflammatory processes of either, whether through continuity of tissue or as a result of the effects of the irritating discharges to which it is exposed. Altogether, the larynx is probably the least protected organ of the system.

Laryngeal stenosis is but the aggravation of a deficiency in the lumen of the vocal organ, a deficiency doubtless necessary for the proper performance of all the functions over which it presides. Its existence begins as soon as by any pathological process the physical conformation of the larynx becomes enlarged. As generally considered, however, the term stenosis obtains when sufficient narrowing of the laryngeal aperture has taken place to interfere seriously with the passage of air.

Etiology.—Among the causes of stenosis in children, those involving an inflammatory process, primarily or secondarily, are by far the most common. The inflammation may either be acute, chronic, or œdematous; it may limit itself to certain parts,—the ventricular bands, the epiglottis, the ary-epiglottic folds, or other portions of the larynx,—or it may involve the entire organ. Probably the simplest form of stenosis is that occurring as a result of a severe attack of simple laryngitis, which is apt to be more serious in children than in adults. Besides the other symptoms present,—*i.e.*, paroxysmal cough, hoarseness, hot and dry skin, frequent pulse, etc.,—a slight dyspnœa, subject in some cases to spasmodic exacerbations, is experienced by the child, whose respiration is decidedly accelerated. A laryngoscopic examination at this time reveals marked tumefaction resulting from extension of the inflammatory process to the submucous tissue, with consequent infiltration of serum loaded with leucocytes. This forms, in our opinion, to all intents and purposes a mild though frequent variety of laryngeal œdema[1] which may suddenly assume a dangerous character.[2] In these cases an additional cause of stenosis may frequently be found in defective muscular action, the result of vascular engorgement or impaired innervation.

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[1] See case of death in undeveloped œdema, quoted by Fagge, vol. i. p. 810, Philadelphia, 1882.

[2] Gottstein, p. 100; translation by McBride, Edinburgh.

True œdema of the larynx, however, is an infrequent cause of stenosis in children, Sestier (quoted by Morell Mackenzie) having found but seventeen cases in children under fifteen years of age in a list of two hundred and fifteen. Its origin may be due to traumatism, impacted foreign bodies, the inhalation of caustics, steam, and other irritating and destructive substances, or it may occur as a complication of eruptive fevers and other general disorders, especially scarlet fever, diphtheria, measles, small-pox, typhoid fever, erysipelas, and pertussis.[1] An interesting case of marked laryngeal dyspnœa occurring as a complication of measles was recently reported by L. Emmett Holt, of New York.[2] At the autopsy the vocal bands were found to be completely destroyed by ulceration, which extended upward to the ventricle and downward about one-fourth of an inch; it had apparently reached the cartilage. The dyspnœa was probably due to œdema or to the presence of a mass of ragged necrotic tissue which was found to cover the laryngeal surfaces. Stenosis of the larynx as a complication of typhoid fever in children was encountered six times in a series of ninety-four cases by Keen, of Philadelphia. In addition to the œdema observed in the course of scarlet fever, an exudation which greatly resembles the pseudo-membrane of diphtheria is often found to act as an effective agent of suffocation. This has also been noticed, though rarely, in measles and in hemorrhagic small-pox. In erysipelas, the œdema is occasionally complicated with a paralysis of the muscles of deglutition which renders easy the passage of food into the larynx.

A stenosis occurring in the course of an inflammatory process may owe its existence to a spasmodic element, even though the inflammation be slight. This is well exemplified by the disease commonly called croup (spasmodic laryngitis, spasm of the glottis), one of the most frequent of the winter disorders to which children in this country are liable. The narrowing of the glottis seems to be due to a disordered action of the excito-motor innervation of the part, the irritant being, in all probability, the slight inflammation of the laryngeal mucous membrane which constitutes the primary element of the disease. Briant[3] called attention to the fact that in malaria attacks of stenosis, resembling those of croup, occasionally occur, in which there is intense redness of the entire respiratory tract.

The action of the inflammatory process in causing the stenosis may be greatly increased by adhesive secretions or false membrane, originating either locally or in neighboring cavities, while, on the other hand, though rarely, these factors may form the only element of obstruction. In the spasmodic stenosis of acute catarrhal laryngitis, for instance, Morell Mackenzie[4] considers it probable "that muscular action operates as a secondary cause, and that it depends primarily on the laryngeal secretion becoming inspissated during sleep, when the mouth is often open. Collecting in this state in the very narrow glottis of the child and adhering to the vocal cords, the thickened mucus gives rise to a gradually-increasing impediment to respiration." This obtains, though to a less degree, in the great majority of affections inducing stenosis, the interference with the expulsive function of the larynx incident to the local disorder doubtless contributing a large share to the retention of the secretions between the swollen surfaces. The manner in which even marked dyspnœa may occur solely through the presence of inspissated secretion was well exemplified by the case of a boy recently seen by the writer, in whom a collection of dry, greenish masses of purulent exudation, almost entirely occluding the glottis, was detected immediately below the vocal bands,—a typical laryngitis sicca. The case was devoid of all hemorrhagic phenomena, however, although these might have appeared had the crusts been roughly removed.

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[1] Gottstein, p. 254; translation by McBride, Edinburgh.

[2] Trans. New York Pathological Society, February 3, 1889.

[3] Gazette des Hôpitaux, No. 40, 1883.

[4] Diseases of the Pharynx, Larynx, and Trachea, English edition.

Diphtheria vividly illustrates the aggravating action of pseudo-membrane upon an already-existing narrowing of the laryngeal aperture occurring as a result of the primary inflammatory process. The false membrane may appear in patches and not occasion much interference with the passage of the air-current; but in the great majority of cases it forms a perfect cast of the laryngeal cavity, and, being comparatively thick and dense, and very adherent to the underlying and generally greatly infiltrated and swollen membrane, it further diminishes the already-reduced calibre of the organ, leaving a small aperture, which may be occluded at any moment by the forcible incursion of a detached piece of membrane from a neighboring part or close of itself through increased infiltration of the underlying cellular tissue.

Though laryngeal syphilis is rare in children, it is nevertheless entitled to consideration as a cause of stenosis in them, the hereditary form, however, appearing to be the only one in which the active ulcerative process or the subsequent cicatricial contraction is sufficiently marked to produce stenosis. In infants even these conditions may prevail, as evinced by Isidor Frankl's case[1] of a child two months old who died of acute stenosis, and whose larynx showed post mortem marked syphilitic disease. Perichondritis and subsequent necrosis form an element of danger in laryngeal syphilis of children, owing to the diminutive size of the surrounding cavities in them and the likelihood of necrosed cartilage falling into the larynx. Cicatricial contraction following active syphilitic inflammation is also a fertile cause of stenosis, as instanced by a case recently reported by Malinowski.[2] An alarming attack of dyspnœa having occurred in a child three years old, in whom the diagnosis of laryngeal syphilis had previously been established by the mirror, tracheotomy was performed, but the trachea was so reduced in diameter, by reason of cicatrices, that the smallest canula could not be introduced. The diagnosis was confirmed by the autopsy.

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[1] Wiener Med. Wochenschrift, 1868, Nos. 69 and 70, quoted by Morell Mackenzie.

[2] Gazeta Lekarska, Nov. 28, 1888; in Journal of Laryngology, etc., April, 1889.

The cicatricial contraction following syphilitic pharyngeal ulceration may also cause considerable interference with the passage of air into the larynx by involving the epiglottis and holding it down over the margin of the aperture. Perichondritis, the result of active specific ulceration, may so aggravate the local infiltration as to greatly compromise the already-narrowed larynx, and seriously endanger the patient in case of a sudden rupture of the pocket of pus, which is usually formed around the inflamed cartilage. The possible escape of the necrosed piece into the larynx is another source of suffocation which may present itself in such cases. J. N. Mackenzie has shown that spasm is a not infrequent complication of congenital syphilitic laryngitis.[1]

In laryngeal phthisis, which occurs in children in the proportion of about two per cent. of all cases, the infiltration, which forms one of the characteristics of the disease, acts as an occasional cause of stenosis. The pathognomonic pyriform swelling of the ary-epiglottic fold, when marked, is usually the first impediment to the passage of air; but serious dyspnœa occurs only when the other portions of the larynx, the ventricular bands, the epiglottis, etc., become œdematous, the swollen parts acting as so many cushions to occlude the laryngeal cavity. This form of stenosis is seldom sufficiently marked, however, to call for surgical interference. Gouguenheim,[2] in an interesting study of the subject read before the Laryngological Section of the International Congress at Copenhagen in 1884, affirms that true œdema of the glottis—or, rather, of the aryteno-epiglottic folds—is exceedingly rare in tubercular laryngitis, and that in almost all cases in which it is present it is due to necrosis of some cartilage. Dyspnœa from this cause is, therefore, of great rarity, since it does not occur in all cases of œdema. With Doléris, he considers the true cause of the tumefaction of the soft tissues to be an infiltration of tuberculous elements.

Another cause of obstruction in this class of cases is the quantity of secretion originating locally and in the lungs, which is discharged with great difficulty, owing to the soreness of the parts, and to their impaired muscular motility, occasionally in itself an element of stenosis. Irregular tuberculous vegetations are not infrequently developed, further restricting the remaining space. Phthisis of the pharynx, by extending to the upper portion of the larynx, may give rise to all the phenomena occurring in connection with the purely laryngeal affection.

Leprosy of the larynx is also a possible cause. An illustrative case was recently reported by Drs. Whipman and Delépine to the Clinical Society of London,[3] of a boy fourteen years of age, in whom death resulted from an attack of dyspnœa which was found to be due to tubercular lupus involving the larynx, trachea, and bronchi. Rachitis is considered by Rhen[1] as a prolific cause of reflex stenosis, that author having never met a case of laryngismus stridulus in which symptoms of rachitis were absent,—a statement which can only be taken with considerable reserve.

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[1] Amer. Jour. Med. Sci., October, 1880.

[2] L'Œdème laryngée dans la Tuberculose du Larynx, vol. iv. p. 122.

[3] British Medical Journal, March 16, 1889.

Laryngeal tumors cause stenosis in almost every case, although the degree of constriction is considerably influenced by the location and size of the growth present. There is considerable variance among writers as to the proportionate predilection of children to laryngeal growths. Taking the tabulations of the principal writers on the subject, however, Fauvel,[2] Mackenzie,[3] Causit,[4] Von Bruns, and others, including thirty-one cases of neoplasms (excluding the cases of tuberculous vegetations which he considers as polypi), collected by Horace Green,[5] and which are generally overlooked, a fair estimate would seem to bring the proportion to at least thirty-five per cent., including congenital cases.

The calibre of a child's larynx being naturally smaller than that of an adult, dyspnœa occurs earlier in the history of the case, the tumor growing as it would in an adult. Another cause for comparatively early stenosis in the majority of infantile cases is the fact that the form of neoplasm most frequently found in children is the multiple papilloma with broad implantation, which in its growth leaves no gap for the passage of air around its margin. A comparatively small neoplasm may give rise to the same result with even more rapidity when situated near the edge of a vocal band and extending along its length, or when situated immediately below or above the anterior commissure, the narrowest part of the laryngeal cavity being thus the first impinged upon. When the growth is pedunculated, the stenosis may be intermittent, especially when sufficiently free in motion to be influenced by the respiratory current, which, either from above or below, according to the location of the tumor, forces the latter between the vocal bands. Position also acts in the same manner, the weight of the neoplasm causing it to locate itself favorably or unfavorably as regards the production of obstruction. A congenital web of membrane between the vocal bands has been met with in a few instances, acting as a cause of stenosis. An interesting case was recently reported by Seiffert and Hoffa,[6] in which the web was so tough that the point of a laryngeal knife was broken when an attempt to incise it was made. In a subsequent report Seiffert stated that he had found the same phenomenon in each of two sisters of the former case, aged twelve and eight respectively.

Foreign bodies of all sorts, of a size permitting their introduction into the oral cavity, have found their way into the larynx, thereby causing more or less obstruction by pure mechanical impediment, by penetrating either or both ventricles or other portions of the cavity, or by pressing upon the epiglottis, which in turn closes partially or completely, as the case may be, the margin of the cavity. Although marked stenosis requires for its production a foreign body of sufficient size to diminish greatly the lumen of the glottis, a very small object may cause serious obstruction by inducing reflex spasm. Again, a diminutive foreign body may endanger life by giving rise to violent inflammation and infiltration, results observed in a case of the writer's,—a young boy, in whom a small sand-burr, which had lain beneath the anterior commissure four hours, had already caused sufficient œdema to interfere alarmingly with respiration.

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[1] Gerhardt, Kinderkrankheiten, Bd. iii. S. 92.

[2] Maladies du Larynx, Paris, 1876.

[3] Essay on Growths in the Larynx, Philadelphia, 1881.

[4] Études sur les Polypes du Larynx.

[5] Surgical Treatment of Polypi of the Larynx, New York, 1859.

[6] Berliner Klin. Wochenschrift, March 5, 1888.

Paralysis of the larynx, especially when the epiglottis is involved, as often seen in the form following or accompanying diphtheria, greatly facilitates the impaction of food or other foreign substances. Another death was lately reported by N. F. Klein,[1] due to strangulation from this cause, the child having been allowed to partake of solid food, contrary to the physician's directions.

Bilateral paralysis of the abductors of the vocal bands, through which the latter are forced to remain in adduction near the median line, is an occasional cause of stenosis in children. Unilateral paralysis—the form most frequently met with in them[2]—may also give rise to a certain degree of interference with breathing, owing to the smallness of the lumen on the healthy side. The sense of suffocation, however, is usually experienced only during physical exercise.

Congenital abnormalities in addition to those already described occasionally give rise to dyspnœa. One of these, which seems to be almost confined to female infants, is a peculiar conformation of the epiglottis, which appears folded upon itself like a leaf on its midrib. This conformation causes its edges to approximate closely the ary-epiglottic folds and to limit greatly the upper lumen of the larynx, producing apparent dyspnœa and a loud and almost constant crowing sound. Other malformations of the epiglottis, the ary-epiglottic folds, etc., have also been recorded.

Obstruction to the passage of air into the larynx may find its origin in disorders of contiguous parts, especially those troubles likely to give rise to œdema,—*i.e.*, diphtheria, tonsillitis, pharyngeal abscess,[3] burns and wounds of the pharynx, etc. Cicatrices resulting from wounds of the larynx and surrounding tissues doubtless produce the same effect as in adults, and are therefore entitled to mention.

Direct pressure may be exerted upon the larynx by swollen glands under the angle of the jaw and cause actual stenosis. In the case of a boy two years of age seen by the writer, for instance, the dyspnœa was so great from this cause that preparations were made for operative procedures, to which, however, it was not necessary to resort. The same effect may be produced by other forms of cervical tumors or by abscesses in the cellular tissue of the neck.

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[1] Polyclinic, December, 1888.

[2] Pointed out by Semon.

[3] Cohen, Diseases of the Throat and Nasal Passages, New York, 1879, p. 245.

Diagnosis.—Important to remember, when a diagnosis in a case of dyspnœa is to be made, is the fact that the function of respiration in infancy and early childhood possesses characteristics of its own, not only in the manner in which it is performed physiologically, but as regards rhythm and proportionate number of functional acts.

Examined during sleep, when disturbing elements will not affect and perhaps modify his nervous equilibrium, a healthy child will be observed to breathe entirely through the nasal passages, which, though narrow, are amply sufficient for the column of air which they are destined to contain. The inspiratory current is alone heard, a gentle, soft souffle, but the expiratory current is noiseless and short and is followed by a brief period of rest. In infants, up to the fourth or fifth week, these acts of respiration do not always alternate regularly, and sometimes present the characters of the Cheyne-Stokes respiration,—*i.e.*, occasional periods of apparent apnœa; but in older children this peculiarity gradually disappears, and the function is finally carried on regularly. In the newly-born the respiration may range from 30 to 45; at six months about 25, and at two years 20.

Another point of importance is the fact that in children of both sexes the respiratory act is carried on principally by the diaphragm and by the lower part of the chest, as in adult males, the ribs moving outwardly to a very slight extent during inspiration. The elevation and descent of the abdominal walls, through the pressure exerted by the diaphragm on the intestines, is, therefore, an excellent guide in ascertaining the movements of both diaphragm and lungs, and of considerable value when compared with those occurring during laryngeal stenosis.

A point of primary importance is whether, though apparently laryngeal, the dyspnœa does not depend upon some interference with the passage of the air-current in some other part of the respiratory tract,—a differentiation which becomes especially difficult when the trachea or the primary or secondary bronchi are involved. In peribronchial adenopathies, for instance, the dyspnœa, which sometimes reaches the stage of orthopnœa, may give rise to laryngo-tracheal whistling sufficiently loud to be heard at a considerable distance, though uncomplicated by paralysis due to pressure on the vagus.[1] As shown by the valuable statistics of H. A. Hare, of Philadelphia, diseases of the mediastinum may present the same source of confusion by giving rise to dyspnœa, which was present in twenty-eight of the forty-six cases reported[1] as occurring in children (sixty per cent.). The thymus gland, instances both of prolonged existence and of excessive growth of which have been reported, may, on account of its position between the sternum and the trachea and of the slight power of resistance of the rings of the latter during infantile life, cause not only dyspnœa but suffocation.[2] Precisely the same statement might be made concerning the thyroid gland, the middle lobe of which, when enlarged, occasionally passes down behind the sternum. Goitre, for instance, and other forms of thyroid tumors, so situated as to be compressed by overlying muscles, occasionally give rise to orthopnœa.

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[1] Jules Simon, Conférences thérapeutiques et cliniques sur les Maladies des Enfants, p. 22, vol. ii., Paris, 1887.

An apparently laryngeal stridor may be due to disorders of the respiratory tract more remote than those described above. In the dyspnœa of bronchial asthma which occasionally follows whooping-cough, measles, or infantile bronchitis, for instance, the distressing wheezing seems to be located at the throat, while in reality it depends upon bronchial stridor, spasmodic or exudative.[3] Emphysema also gives rise to the same peculiarity in a large proportion of cases.

While the laryngeal mirror, revealing at once the unimpaired motility and practically normal appearance of the larynx, might alone be sufficient to establish beyond a doubt the peripheral cause of dyspnœa in these cases, the difficulty often encountered in using the instrument satisfactorily in children renders the diagnosis doubtful in a proportionate number of cases, unless other means of diagnosis at our disposal be carefully employed. This may, in fact, be said of all disorders of childhood in which the laryngeal mirror has to be employed, especially when the cases are seen in their incipiency and when febrile symptoms are not present as an element of the disorder. Many of these means even are sometimes likely to mislead. The character of the voice, for instance, considered by some authors as a valuable differential sign, loses much of its value when we take into consideration that in the pulmonary disorders capable of presenting apparently laryngeal dyspnœa as a symptom hoarseness is not infrequently present. Fremitus over the seat of constriction, which a sensitive touch can detect readily and to great advantage, may also be simulated by the presence of mucus, which conveys to the finger the same impression as a constriction,—itself, in fact, often the seat of an accumulation of mucus. The appearance and movements of the chest, even, may be of no diagnostic value, since many of the disorders, whether laryngeal, tracheal, or bronchial (primary ramifications), present in this particular the same clinical picture. We have, however, in auscultation and percussion almost unerring means to settle any mooted question.

By means of the first, the location of the obstruction can be ascertained with certainty, at least as far as the second bifurcation. The peculiar wheezing sound (*cornage*) which is always an element of the case when constriction of either the larynx, the trachea, or the larger bronchi is present,[1] and which heard from a distance seems generally to be located at the larynx, does not convey the same impression, especially when the stethoscope is used ; but the wheezing sound may be traced down along the trachea, which acts as a resonant chamber, to the seat of obstruction, where it stops, to give place further on to sounds varying according to the local disturbance, but differing completely from that heard above. Located at the larynx it is readily detected, the sounds varying with the mechanical cause of the constriction, the density of the parts, and the presence or absence of secretions. Though transmitted to surrounding cavities, the sound gradually diminishes as the distance from the larynx is increased, its greatest intensity, as in the case of peripheral constrictions, being located at the stenosed area. Percussion is of value by giving us an idea of the relative proportion of air between the parts of the respiratory tract above and below the constriction,—decrease in the normal proportion of air being manifested by dulness, and increase by exaggerated resonance. When the stenosis is at the larynx, and is of a character inducing inspiratory dyspnœa, the normal quantity of air in the chest is naturally reduced, and dulness results. Expiratory dyspnœa, on the contrary, causes the chest to be overfilled, thus giving rise to increased resonance, sufficiently marked at times to resemble tympany. In constrictions below the larynx the percussion-note remains the same until the stricture is reached, when suddenly a small area of dulness is observed, which is either continued, or replaced by exaggerated resonance, as the case may be. The other symptoms are naturally necessary to determine the individual nature of each case.

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[1] The Pathology, Clinical History, and Diagnosis of Affections of the Mediastinum, including the Clinical History of Five Hundred and Twenty Cases, Philadelphia, 1889.

[2] Quain's Dictionary of Medicine, New York, 1887, p. 1630.

[3] T. B. Berkart, Bronchial Asthma, 2d ed., London, 1889.

The differential diagnosis of stenosis occurring as a complication of inflammatory disorders of the throat or of eruptive fevers does not require to be dwelt upon, a proper recognition of the primary affection determining at the same time the cause of the constriction and its nature. More difficult to diagnose, however, are the cases in which no febrile elements exist, and in which alone the symptoms resulting from stenosis are present.

In each of these diseases a positive diagnosis can be determined by the laryngoscope, in some cases with, in others without, the assistance of the general symptoms. Without the mirror, however, all must remain within the domain of uncertainty, a fact which should cause the practitioner to spare no effort to obtain a careful examination, even if it is necessary to resort to anæsthesia or to the artificial elevation of the epiglottis.

As it is not intended that this article should comprise individual affections of the larynx in relation to differential diagnosis, treatment, etc., the reader is referred to the portions of the work in which the several diseases are described.

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[1] Dieulafoy, Manuel de Pathologie interne, tome i., Paris, 1888.

TUMORS OF THE LARYNX.

By SIR MORELL MACKENZIE, M.D. Lond.

BENIGN GROWTHS.

The statistics given by different authorities on this subject do not agree as to the relative frequency of benign laryngeal growths in children. In 1854 Middeldorpf published sixty-four cases of tumor of the larynx, but the age of the patient is mentioned in only twenty-nine; of these, eight, or 27.5 per cent., were children. Of thirty-one cases of laryngeal growth collected by Gibb, thirteen, or 41.6 per cent., were children, and in eight of these the tumors were supposed to be congenital. Causit[1] found that, in forty-six cases gathered from various sources in which the age was recorded, ten were classed as congenital, seven were between birth and the age of two years, and twenty-six were between the ages of two and twelve. He considers that laryngeal growths are more common in infancy than at any other period of life. Von Bruns gives[2] one hundred and twenty-seven cases of papilloma in children, of whom ninety-nine were under fifteen years of age. On the other hand, Fauvel[3] among three hundred cases under his own care met with only five under the age of ten, and seven between ten and twenty. My own experience, as recorded in my book "Growths in the Larynx," published in 1871, showed that in one hundred consecutive cases in only two instances were the patients under the age of five years, in four between five and ten, and in four between ten and fifteen. This shows a percentage of ten patients under the age of fifteen years.

The table on the following page gives the details of thirty-four cases of laryngeal growths in children, observed since the publication of the above work. These are taken from my notes of over four hundred cases of laryngeal growths occurring both in children and in adults, and the percentage of children affected with growths would therefore appear to be somewhat less than that shown by the previous collection published in the work just referred to. I am inclined to think, however, that the proportion of children who suffer from laryngeal growths is much higher than has hitherto been supposed, and that Kohler and Lewin are probably right in their opinion that in nearly half of the cases of laryngeal growths met with the patients will be found to be children. It is extremely probable that many cases of growths in infancy and childhood are overlooked, or that the symptoms to which they give rise are ascribed to other causes by physicians who do not use the laryngoscope; moreover, many growths may escape detection, even with the laryngoscope, on account of the great difficulty in examining children and of the more pendent position of the epiglottis in them.

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[1] Études sur les Polypes du Larynx, p. 10.

[2] Die Laryngotomie.

[3] Maladies du Larynx, 1876, p. 197.

SEX AND AGE.	Cured by endo-laryngeal treatment.	Cured by endo-laryngeal treatment after tracheotomy.	Incomplete cures.	Died from tracheotomy.	Lost sight of.
Males:					
2 at 4 years old	1	. .	. .	. .	1
3 " 5 " "	1	. .	1	1	. .
5 " 6 " "	2	1	. .	. .	2
3 " 7 " "	1	. .	2	. .	. .
6 " 8 " "	2	. .	2	. .	2
1 " 9 " "	1	. .	. .	. .	. .
1 " 10 " "	1	. .	. .	. .	. .
21	9	1	5	1	5
Females:					
1 at 4 years old	1	. .	. .	. .	. .
2 " 5 " "	. .	1	. .	1	. .
1 " 6 " "	1	. .	. .	. .	. .
3 " 7 " "	2	. .	. .	1	. .
5 " 8 " "	1	. .	2	. .	2
1 " 9 " "	1	. .	. .	. .	. .
13	6	1	2	2	2
Summary:					
Males	9	1	5	1	5
Females	6	1	2	2	2
	15	2	7	3	7[1]

As regards *congenital* laryngeal growths, up to 1871, when I published the work already referred to, such a condition had been proved to exist in only five cases,—viz., two reported by Defour, one by Dr. Arthur Edis, and two others which occurred in my own practice. The case of Dr. Edis is further remarkable inasmuch as the tumor was a cyst which caused death by asphyxia thirty-seven hours after birth. Since then, however, Von Bruns has published, among his statistics, records of twenty-three cases of congenital tumors of the larynx. Four of the thirty-four cases mentioned above were presumably congenital, as aphonia had existed from birth.

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[1] I have since learned that three of these cases died,—two from suffocation, and one from pneumonia. No attempt had been made to treat the condition in the larynx.

The variety of benign growths most frequently found in children is papilloma. In my thirty-four cases, twenty-nine patients had papillomata and five were of fibrous structure. Though fibromata are, as is seen, occasionally met with, myxomata are extremely rare. I have observed only one case. I know of no instance of angioma having been found in a child's larynx. Cysts are usually found situated on the epiglottis, but may sometimes be seen in the larynx.

Etiology.—The factors, apart from age, which make up the etiology of laryngeal growths in children may be divided into predisposing and exciting. Among the former are sex, hereditary influence, constitutional peculiarity, the conditions of life and surroundings, and the influence of acute diseases.

The influence of sex has not been fully investigated, but the generally-received opinion is that laryngeal growths are more common in males. In the above thirty-four cases there were twenty-one boys and thirteen girls. Von Bruns (*op. cit.*) found that, of one hundred and thirteen children affected with laryngeal growths, seventy-three were boys and forty girls; and Causit, who has given more attention to this subject, says that the proportion of males to females in his cases was as twenty-eight to fourteen.[1]

Heredity does not appear to have any special influence on the production of these growths. Poyet, however,[2] met with papillomata of the larynx in a brother and sister whose father was stated to have been similarly affected, and he afterwards treated two brothers for the same disease.

Constitutional peculiarity may have some slight influence. I do not, however, believe that hereditary syphilis or tuberculosis predisposes to the formation of true growths, though these diseases may produce false or inflammatory excrescences. A curious case is recorded by Hering, in which a man aged thirty-three, who had multiple fibromata on his skin from birth, had also a tumor of a similar nature in his larynx which necessitated tracheotomy. A remarkable fact in the history of this case was that his mother had been similarly affected. Poyet also states (*loc. cit.*) that the brother and sister referred to above had warts on their hands, a condition which he states he has "very frequently" found in persons suffering from papilloma of the larynx. I have myself twice met with warts on the fingers in the case of children suffering from laryngeal excrescences.

The conditions of life and surroundings appear to have a similar influence in the causation of laryngeal growths as in that of other diseases of the respiratory passages. A large majority of the cases will be found among the children of the poor, whose conditions of life leave them more exposed to the action of the immediate causes of these affections.

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[1] Op. cit., p. 12.

[2] Manuel pratique de Laryngoscopie et de Laryngologie, Paris, 1883, p. 313.

The influence of acute diseases, such as measles, scarlatina, etc., is exerted indirectly through the chronic catarrh which they may leave as a sequel. Causit considers that the pathogenic action of these diseases is by no means proved, and that the occurrence of laryngeal tumors after an acute disease is to be looked on as *post hoc* and not *propter hoc*.

The exciting cause of the development of growths in this situation is chronic irritation of the laryngeal mucous membrane, whereby a chronic catarrhal condition is induced. All the other so-called exciting causes—over-use of the voice, exposure to cold, inhalation of irritating particles or vapors—act by causing in the first instance chronic catarrh of the larynx. Repeated attacks of post-nasal catarrh and pharyngitis also produce this state of continued hyperæmia of the larynx, probably by the irritating nature of the secretions that are constantly trickling into it. This chronic congestion is aggravated by the hawking and coughing induced thereby. Bearing in mind the presence of a constant irritant of a more or less intense nature, we can easily follow the stages of the development of a laryngeal tumor, from irritation and hyperæmia to gradual thickening of the mucous membrane, with proliferation of certain groups of cells and the ultimate formation of growths of various kinds. At the same time, it is almost impossible to assign a definite proximate cause for the presence of such a tumor in the larynx of any individual patient, as its growth is so slow that symptoms are often not produced until it has attained a considerable size, and it may therefore have existed a long time before the cause to which its presence is attributed had begun to act.

Symptoms.—The symptoms will depend, as in adults, upon the size, situation, structure, and rate of growth of the tumor. The most constant symptom is an *alteration in the character of the voice*, varying from slight hoarseness to complete aphonia. According to my own experience, hoarseness or aphonia existed in ninety-two per cent. of all cases of laryngeal growth. I have found the percentage about the same in my thirty-four children's cases, as the voice was altered or lost in thirty-one instances, or ninety-one per cent. Of these thirty-one cases, there had never been any voice in four instances, the children never having cried out loud or made any sound of any kind; in seven cases there had been "something wrong with the voice," the children having cried in a peculiar manner; in eighteen of the patients the voice had become affected between the second and the third year, in one case at the fourth, and in another at the eighth year of age.

Causit[1] says that this symptom was absent in only five of the cases collected by him. In fifty-two per cent. of my cases this change in the voice was the only symptom. When the laryngoscope can be used the growth can be seen, but in very young children this mode of examination is often very difficult, or even impossible. It is often, also, almost impossible, especially if the parents be ignorant, to ascertain whether the child has ever "sounded its voice." Such people are apt to confound phonation with articulation, and, though a child may cry loudly, they think that, as it does not speak, it has no voice. In such cases careful inquiry as to whether the child has cried when hurt or hungry will often settle the question. The possibility of the existence of deaf-mutism (especially in infants), paresis or paralysis of the vocal cords from disease of the central nervous system, anomalies of development, and the influence of reflex irritation should all be excluded. Among the anomalies of development is a very rare one which I have met with three or four times. The apices of the arytenoid cartilages and the cartilages of Santorini were so long that they prevented the approximation of the vocal cords. In one of these cases, in which I had an opportunity of seeing the patient again when he had reached adult life, the hypertrophied parts had, so to speak, attempted to adapt themselves to circumstances by overlapping, so as to allow the cords to approach each other: in this way a hoarse but intelligible voice was produced. As an instance of aphonia due to reflex irritation, I may mention a case reported in the *British Medical Journal*, March, 1887, by Dr. W. G. Walford, in which a boy who was suffering at the same time from colic and aphonia recovered his voice when the colic was relieved. The degree of aphonia bears no relation to the size of the growth, as a very small sessile growth on the cord itself may interfere with its functions more than a large one, which often becomes pedunculated, and, being forced up out of the glottis by the current of air, interferes very little with the production of sound (Czermak). On the other hand, subglottic growths, by being forced into the glottis during phonation, often cause aphonia. The intermittence or sudden occurrence of aphonia is sometimes presumptive evidence of a subglottic growth.

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[1] Op. cit., p. 18.

Cough, according to Causit, is rather a frequent concomitant of laryngeal growths in children. It occurred in twenty-three out of his forty-six cases. It usually comes on in paroxysms, and, if the child is old enough, it may complain of a tickling sensation in the throat which precedes and excites the cough. The cough itself is usually croupy, and is accompanied by expectoration of mucus when there is any considerable degree of catarrh.

Dyspnœa is a very common symptom, especially in infants. The difficulty in breathing is often paroxysmal, and is generally worse in damp weather,—a fact which leads me to think that the growth may by absorption of moisture become larger and encroach upon the aperture of the glottis more than usual. Causit remarks that the paroxysms are apt to come on in the night, especially if the child is very young.

Pain.—There seldom appears to be any pain in the cases of benign laryngeal growth. If the little patient is old enough to give any account of its symptoms, it will be more likely to complain of the tickling sensation already described in connection with the cough than of any actual pain.

Dysphagia.—I have not met with any cases in children in which there was any difficulty in swallowing.

With regard to the *general symptoms* caused by tumors of the larynx, in the early stages very little effect is produced on the general health, but, as the growth increases in size, the interference with respiration will generally cause anæmia and malnutrition, and the child will become pale, thin, and fretful.

Diagnosis.—When it is possible to get a good view of the glottis, the evidence of the laryngoscope is, of course, conclusive. Should the laryngoscopic examination be impossible or unsatisfactory, the following aids to diagnosis may be used.

Forcible depression of the tongue will occasionally permit a view of growths connected with the epiglottis. If the larynx be raised by grasping the thyroid cartilage, at the same time that traction is made on the tongue, the upper orifice of the larynx may sometimes be inspected.

Examination with the index finger may give some idea as to the position, size, form, and consistence of tumors situated above the vocal cords. The value of the results will of course depend very much on the tactile delicacy of the physician and on his experience in this mode of examination. It should be employed with great caution, as any roughness may bring on an attack of dyspnœa which may even be fatal in some cases.

Auscultation of the larynx is seldom of any value in children: at the most, it gives but the signs of laryngeal obstruction. Causit (*op. cit.*), however, says that a sibilant sound in the larynx on inspiration, heard especially at night, is rarely absent in children suffering from laryngeal growths. Very small tumors do not, however, modify the respiration in any way.

Expectoration of fragments of the tumor is very liable to occur in cases of papilloma. If aphonia is also present, the expulsion of such fragments may be taken as presumptive evidence of the existence of a growth near or upon the vocal cords.

When the presence of a growth has been ascertained, the next step is, if possible, to determine its nature. In describing the laryngoscopic appearances, I shall follow the method adopted in my previous works, and shall separate the different tumors according to their pathological character.

Papillomata are most frequently situated at the anterior commissure of the vocal cords; sometimes, as in a case in my own practice, they may form a papillomatous membrane uniting the cords for a considerable extent. They are also found on the ventricular bands or on the epiglottis. They are often multiple, and sometimes they occupy a symmetrical position on both sides. Sometimes they are large, reddish, cauliflower excrescences; this variety is the most serious, on account of its liability to recur. The other varieties are usually pink, but may be dark red or grayish. Their size varies from that of a mustard-seed to that of a large pea, but they seldom attain very large dimensions in children.

Fibromata appear as round or oval bodies situated on the vocal cords. They are not often met with at the anterior commissure, and are comparatively rare in other situations. They are generally pedunculated and solitary. The surface is usually smooth, but may be irregular. In color they are usually of a rather bright red, but they may be pink or grayish. They are more commonly rather hard and firm, but if they are soft they are likely to become œdematous, or they may become ulcerated and give rise to hemorrhage, which may be severe, as some of these growths are very vascular.

Myxomata are extremely rare. I have met with only one case: the tumor, which was pink and transparent as seen with the laryngoscope, was only partly mucous.

Cysts occur most frequently on the anterior surface of the epiglottis, but may also be situated on the vocal cords or ventricular bands. They are rarely larger than a pea, and are sometimes pedunculated. Their color is generally red, and, as they cause irritation, they are surrounded by a hyperæmic zone. Schwartz[1] mentions a case of Krakauer's in which a cyst as large as a hazel-nut occurred on the left ary-epiglottic fold in a boy aged ten. It was removed by subhyoid pharyngotomy. He also mentions a case reported by Blanc, in which a cyst as large as an almond grew from the left sacculus laryngis in a girl of ten, pushed the epiglottis upward and to the right, and obliterated the glottis for four-fifths of its extent. From the nature of its contents, it was believed to be a dermoid cyst of the third branchial cleft, which had pushed its way into the larynx. Symptoms of dyspnœa had existed since birth. There had been a small abscess on the side of the neck, the opening of which had relieved the breathing. This was probably part of the cyst which had become cut off from the rest.

I am not aware of any case of angioma having been found in a child. I have met with only two cases in adults: the growths had a blackberry appearance, and were situated in one case in the right hyoid fossa, in the other on the right ventricular band.

MALIGNANT GROWTHS.

The occurrence of malignant growths in children is so rare as to be little more than a pathological curiosity. As, however, one or two undoubted cases have been recorded, the possibility of such an event should be borne in mind.

Epitheliomata.—An example of this class has been positively proved to have existed in a child aged three years.[2] A secondary deposit was found in one of the glands of the neck.

Sarcomata.—One case has been reported by Gottstein in which a fibrosarcoma was situated at the anterior commissure. Sarcomata present almost the same laryngoscopic appearances as papillomata. They are generally smooth, but may be mammillated. The color is usually red, but may be yellowish.

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[1] Tumeurs du Larynx, p. 37.

[2] Rehn, Virchow's Archiv, vol. xliii p 129, 1868.

Prognosis.—In children the prognosis is always more serious the younger the patient. This is due to the small size of the larynx, and to the great difficulty attending the diagnosis and treatment. Moreover, other laryngeal affections—such as acute inflammation of the whole organ, excoriation of neighboring parts from rubbing against the growth, œdema glottidis, bronchitis, and pneumonia (by extension of irritation)—are more likely to attack children suffering from growths. The prognosis is also unfavorable from the fact, pointed out by Schwartz,[1] that papillomata—the variety of growth generally met with in children—have a marked tendency to recur and often become multiple. Again, surgical treatment, more especially tracheotomy, is more dangerous in children than in adults. As will be seen from the table given on page 506, the results of endo-laryngeal treatment render the prognosis more hopeful in cases in which it can be employed. Of twenty-two cases operated on in this manner, all the patients recovered from the operation, and in only two cases was the growth not completely eradicated. The results of tracheotomy followed by endo-laryngeal removal of the growth were by no means so favorable, as of five patients who underwent this treatment only two recovered: the others died, one during the operation, one from exhaustion in forty-eight hours, and one in three or four days from pneumonia.

Treatment.—This may be palliative or radical.

The only safe *palliative treatment* is the timely performance of tracheotomy, especially if the child's respiration is at all embarrassed. I have found that this treatment is usually attended with the best results; and Von Bruns[2] records a similar experience.

Radical treatment may be either endo-laryngeal or extra-laryngeal, or both these methods may be combined, as in cases in which a preliminary tracheotomy has to be performed for the safety of the patient and the tumor is afterwards removed through the mouth.

Endo-laryngeal treatment is either mechanical or chemical, and does not differ in any respect from that in the adult except in the greater difficulty of applying it. The younger the child, the less easy, of course, it is to treat, but it will always be found that a certain number of children, even under five years of age, can be successfully operated on. Von Bruns has reported nineteen cases of endo-laryngeal operations on children under ten years, three of the patients being less than four years old. Recurrence took place in only one case. In addition to six cases of growth in children which among others I treated successfully by endo-laryngeal means, and of which details were published in my book "Growths in the Larynx," I have operated successfully in seventeen of the thirty-four cases since collected. In most of these operations I used my tube-forceps, but latterly I have almost exclusively used my rectangular forceps, made rather more delicately than those employed in the case of adults. In two of these cases a preliminary tracheotomy was performed, the growths having been subglottic in position.

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[1] Tumeurs du Larynx, p. 77.

[2] Die Laryngotomie.

Mechanical treatment is carried out either by evulsion, crushing, or cutting. Anæsthesia should, if possible, be produced by means of a five-per-cent. solution of the hydrochlorate of cocaine. Chloroform narcosis is useless, as endo-laryngeal operations cannot, as a rule, be carried out under its influence unless a preliminary tracheotomy has been performed. If the symptoms are not urgent, it will greatly facilitate the operation if such conditions as congestion of the fauces, hypertrophy of the uvula, or enlarged tonsils be subdued by appropriate treatment. Congestion of the larynx, if at all marked, must be relieved, as while it exists any operation would be likely to increase it and thereby endanger the life of the patient. All instruments should, of course, be warmed before being introduced.

Evulsion is performed by means of suitably-curved forceps. I usually operate with my own rectangular forceps (antero-posterior), but I have used the tube-forceps in a large number of cases with satisfactory results. The use of the tube-forceps is not, however, free from danger. I have known a case in which the inner stem broke and one of the claws was left in the larynx. It was fortunately coughed up two or three days later, and no ill consequences followed; but such an accident might easily be fatal both to the patient's life and to the surgeon's professional reputation. Evulsion is most suitable in the case of sessile growths, but all kinds of growths except cysts may be removed in this way. Râclage or grattage, a form of evulsion recommended by Voltolini, is really a revival, or rather an adaptation, of the treatment of nasal polypi described by Hippocrates. It is performed by means of a rather rough piece of sponge firmly attached to a suitably-curved stem. The sponge is moved rapidly up and down over the site of the growth, the latter being thus torn off. I have found it useful in the case of small multiple growths, and it is especially valuable in the case of children who do not tolerate the laryngeal mirror. The chief objection to this method is the danger of fragments falling down the trachea.

Crushing is performed by means of the same forceps as are used for evulsion.

Cutting operations, if performed on children, should always be done by means of cutting-forceps or guillotines, as the introduction of laryngeal knives or lancets, as recommended by Tobold in the case of adults, is attended with too much danger. In the case of cysts, however, it is better to puncture the cyst than to tear it away with forceps. The evacuation of the contents is generally sufficient to effect a cure.

Caustics, if used at all, must be very concentrated, and should be applied only to the diseased tissue. The difficulties attending their use and the unsatisfactory results obtained thereby have led to their almost entire abandonment as a means of treating these affections.

Extra-laryngeal treatment is seldom called for unless in the case of very young children whom it has been found impossible to treat by endo-laryngeal methods. It is never indicated unless life is threatened by dyspnœa, and in many cases a combination of tracheotomy with endo-laryngeal treatment will be found sufficient. The immediate danger to life, and the almost certain destruction of the voice should the patient happen to recover, render laryngotomy an unjustifiable operation unless as a last resource in impending suffocation.

The extra-laryngeal operations are the following: (1) thyrotomy, or division of the thyroid cartilage; (2) supra-thyroid laryngotomy, in which the incision is made through the thyro-hyoid membrane; (3) infra-thyroid laryngotomy,—*i.e.*, through the crico-thyroid membrane; (4) tracheotomy, either as a palliative or as a preliminary to other measures. As an antecedent measure to thyrotomy, it should, if possible, be avoided; but it may become necessary if dyspnœa is present. If tracheotomy be done, endo-laryngeal means should be tried before recourse is had to the capital operation as a last resource.

In performing *thyrotomy*, the incision should be made exactly in the middle line from the thyroid notch to the upper border of the cricoid cartilage. In dividing the thyroid cartilage, its upper angle should, if possible, be left intact, as in this way, after the wound has healed, the relations of the vocal cords to each other are not disturbed and there is less risk of permanent aphonia. The alæ should then be gently drawn aside with retractors held by two assistants, one on each side of the patient. If the alæ cannot be drawn back sufficiently to allow of the easy removal of the growth, the crico-thyroid membrane should be divided along the lower border of the thyroid cartilage. If this does not give room enough, the upper angle of the thyroid cartilage should be cut through, or even the thyro-hyoid membrane must be divided along the upper border of the thyroid cartilage. Division of the cricoid cartilage does not facilitate the removal of the growth. A strong light should now be thrown into the larynx from a frontal mirror. The growth should be seized with forceps and cut off with curved scissors. Its base should then be touched with a strong solution of nitrate of silver, or with the galvano-cautery at a dull heat, so as to arrest bleeding. The alæ should then be brought together as nearly as possible in their normal position, and fixed by two silver sutures. The wound in the skin may be sutured or united with plaster.

In the case of subglottic growths it is unnecessary to divide the cricoid cartilage, as they can be reached through the crico-thyroid membrane, or through an opening in the trachea.

Von Bruns has collected seventeen cases of thyrotomy performed on children. Eight cures were effected and nine recurrences were reported.

Supra-thyroid laryngotomy may be performed in the case of large growths in the upper part of the larynx which cannot be removed through the mouth. A transverse incision should be made along the lower border of the hyoid bone, through the skin, the fascia, the inner halves of the sterno-mastoid muscles, the thyro-hyoid membrane, and the glosso-epiglottic fold or ligament. The epiglottis should be seized on one side and drawn through the wound. The growth may then be removed by the same means as in thyrotomy. This operation is by no means so dangerous to life as thyrotomy, and it is not so likely to cause permanent injury of the voice. At the same time, I can scarcely consider this operation necessary, as in the cases in which it would be most suitable the growth can generally be easily reached and removed with forceps through the mouth.

Infra-thyroid laryngotomy is sometimes applicable in the case of infra-glottic growths or of those situated on the lower surfaces of the vocal cords. Instead of merely making an incision in the middle line and opening the crico-thyroid membrane, as in ordinary crico-thyroid laryngotomy, it is better in the case of growths to dissect away the superficial structures covering the membrane to such an extent that the opening therein is fully exposed. A canula should then be inserted and allowed to remain until any tenderness and tendency to bleed which may ensue have passed away. After two or three days the canula may be removed, and the exact site of the growth may be determined by means of a small infra-glottic mirror passed through the opening. The examination having been made, the mirror is laid aside, and the growth is removed by means of suitable forceps. This operation is not practicable in the case of very young children, on account of the extremely small size of the crico-thyroid space.

After the removal of growths by infra-thyroid laryngotomy, the canula should be worn for some weeks, or even months, in case recurrence should take place.

SPASMODIC LARYNGITIS.

By WILLIAM P. NORTHRUP, M.D.

Synonymes.—Spasmodic croup, False croup, Catarrhal croup.

Etiology.—*Age.*—During first dentition, and especially in the second year of life, catarrhal croup is most frequent. It is often met with in the third and fourth years, occasionally in the fifth, less frequently in the sixth and seventh years, but in individual cases may persist till the fifteenth or sixteenth.

Sex.—It is said to occur more frequently in male children than in female.

Epidemics and Seasons.—Epidemics of measles and scarlet fever, seasons favorable to catarrhal inflammations, coryza, and bronchitis, furnish the greatest amount of laryngitis and in children catarrhal croup. But few occur in summer, the cold, damp months of winter being particularly favorable to its development. It often occurs among the early symptoms of whooping-cough and measles.

Constitutional.—It has been asserted by some teachers that vigorous children have shown a more marked tendency to this malady than feeble ones. In individuals and in families the tendency to recurrence of catarrhal croup has persisted till the age of puberty.

Determining Causes.—Gastric catarrh, indigestion, and associated catarrhal inflammations have been the exciting cause of croupal attacks. Among the mechanical causes may be mentioned screaming, violent coughing, inhalations of irritating vapors, hot steam, hot smoke, dust, and cold air, sudden chilling of a portion of the body, exposure to damp and cold.

In addition to cases occurring with marked and sufficient local lesions, which are easily studied and verified, there are cases of moderate laryngitis with a spasmodic element, which so far has been explained in no better way than by calling it reflex spasm. The irritation is furnished in the mucous membrane of the larynx. The return stimulus induces convulsive seizures in the laryngeal constrictor muscles, and this it is which transforms a mild "cold and sore throat" into a condition of spasmodic croup.

Pathology.—The child's larynx is relatively small in capacity compared to that of adults. Added to the narrowness of the glottis is the condition of loose and vascular mucous membrane, which resembles that of the bronchial tubes. Congestion and œdema enlarge the cords into valve-like bodies, which coarsely vibrate on inspiration and part on forced explosive expiration, giving the characteristic barking cough.

The mucous membrane, at first red, varying to violet, is commonly dry, and shows a uniform lesion involving the entire extent of the larynx and extending to the trachea. It may, however, in mild cases, be limited to the epiglottis, aryteno-epiglottic folds, or false and true cords.

This dry state gives place to a moist, flabby condition of the portions most involved, the mucous membrane becoming coated with tenacious viscid mucus. Later pus appears in the products of inflammation, which gives to the sputum of older children the nummulated, yellowish characteristics which mark the last or "loose" stage. The inflammation is ordinarily superficial, being limited to the mucous membrane. Its severity may, however, become such as to cause destruction of superficial tissues and result in shallow ulcers. These may be upon any portion of the larynx or epiglottis. They have been frequently observed at the anterior junction of the cords, less often upon the cords, and occasionally at their posterior attachments. It is not common for more than one ulcer to be present. This lesion may easily escape detection in a casual examination, and it is recommended to float the organ in water for the better demonstration of slight superficial loss of substance.

After death the elasticity of the submucous tissue of the larynx may cause the swelling in part to disappear, and leave the membrane wrinkled and pale. So, too, œdema may disappear, and a patient who in life gave evidence of distressing dyspnœa and unmistakable obstruction may at autopsy show but the shrivelled mucous membrane which had covered a swollen and œdematous false cord and aryteno-epiglottic fold.

O'Dwyer maintains that obstructive swelling is located in the narrowest part of the air-passage,—viz., within the cricoid cartilage. The mucous membrane is confined within the calibre of a resisting ring, and any swelling it may take on necessarily causes it to encroach upon the capacity of the air-passage. This swelling, he claims, does not disappear at death, but can be demonstrated by a horizontal cut through the cricoid cartilage and mucous membrane. The swelling of the folds and false cords is not likely to produce stenosis, in croup, before the much narrower passage of the chink and subglottis shall have already given rise to symptoms of obstruction. Occasionally severe dyspnœa, requiring intubation, has existed, and yet the voice has continued clear. In such cases the stenosis has been subglottic. Petechiæ may be among the permanent remains of an intense inflammation.

Symptoms and Course.—Acute catarrhal laryngitis of mild type begins with hoarseness, followed by aphonia which may alternate with hoarseness, often with marked febrile movement. The child continues to play about. A hoarse cough may be the first announcement of the beginning of the malady. During the day a moderate flushing of the face and heat of the hands may attract attention, but there is no stridor to respiration, and no change of facial expression. These symptoms may become marked during the following night, and on the second and third nights may be even worse, and the case go on thereafter to speedy recovery without having excited grave apprehension.

The most common picture of laryngitis with spasm presented to the mind of the practising physician is of a mild case, in which during the day the child has coughed a little hoarsely, without feeling ill. At nightfall the cough has been observed to be a little "tight." The child goes to sleep quietly without noticeable fever and without anything to attract the attention. After a short sleep he awakes suddenly with great oppression of the chest. Inspiration is prolonged, stridulous, and crowing, followed by a short, explosive, barking cough. The child becomes frightened, wishes to be taken up, clutches at the attendant's garments or face, climbs upon her shoulder, and manifests great restlessness and distress of mind. He tries to cry out or to speak, and his vocal cords refuse to vibrate, and only the coarse flutter of mucus or swollen folds of membrane respond and add to the fright of the child.

Such attacks pass off after the usual exhibition of domestic remedies or after the child has cried and coughed. This easy relief from severe symptoms suggests the explanation that dried, tenacious mucus collected upon the vocal cords during sleep is probably the cause of the muscular spasm. The onset was sudden and severe, and relief came promptly. It is usual for the child to fall asleep again and finish the night with moderate restlessness and coughing, or awake with another severe attack and again sleep tranquilly in early morning. During the day he seems nearly or quite well, and on the following two nights and intervening day repeats the cycle described above,—viz., comfortable days, alarming attacks at night. These symptoms usually extend over three to five nights and as many days and end in recovery.

In cases arising from trauma the lesion may be of the nature of severe catarrhal inflammation, or the superficial epithelium may be destroyed. The severest forms of laryngitis are met with among the poor, and are due to the attempt of the child to drink from the spout of a teakettle, thereby inhaling hot steam. In the cases which go forward to recovery, the fever subsides with the dyspnœa at an early date, stridulous respiration disappears, aphonia gives place to an intermittent hoarse voice, with hoarse cough, expectoration becomes muco-purulent and abundant, coarse tracheal râles announce a similar process taking place in that region, and at the same time the patient has a free nasal discharge. The last-named symptom has been regarded in German domestic circles as an assurance that the disease is progressing favorably, and has given rise to the salutation after sneezing, *So, Gesundheit!*

Diagnosis.—Catarrhal laryngitis with cough and dyspnœa may be confused with pneumonia with grunting respiration. Occasionally operators who are called to perform tracheotomy or intubation find a case of pneumonia awaiting them. A sufficiently careful examination of the chest will answer the inquiry. Recessions, supra-sternal and supra-clavicular, belong to laryngeal obstruction, and are well marked and unmistakable. These are wanting in pneumonia.

In traumatic laryngitis, as from inhalation of hot steam, inspection of the lips, mouth, and fauces, and digital examination of the epiglottis and mucous folds, may furnish evidence of the injury in the larynx. It is quite common in New York, among a certain class, for the mother to lock her young children in her tenement-rooms to go for a short errand to the stores. Among the accidents of her absence, not infrequent are burns and scalds and hot-steam inhalations. It is desirable to remember, too, that the symptoms may follow the injury after the lapse of several hours.

Prognosis.—Death from uncomplicated spasmodic laryngitis is extremely rare.

Treatment.—*Prophylaxis.*—It is desirable to habituate children to the out-door atmosphere in the sunniest and dryest part of the day in selected days of the week. In patients subject to croup it is believed that dry air, even quite cold, is beneficial in its effect upon the mucous membrane. Judicious use of sponge-baths and rubbing with the bare hand over the larynx render the skin more insensitive and less susceptible to the harmful action of cold air.

Causal Indications.—Many children bring about a congested and irritable condition of the larynx from excessive screaming. Among older children, at games, the practice of screaming, at the same time exercising violently and inhaling over the susceptible vocal cords cold air or cold and moist air, gives rise to laryngitis which may be attended with spasmodic croup or aphonia. These practices, coupled with exposure of the limbs and feet to cold and dampness, bring together several potent factors towards catarrhal conditions and croup as met with in every-day practice. Children who are taken to street-corners to wait in the open air to witness parades and public demonstrations are sure to furnish a contingent in professional practice in the following few days. By meeting these causal indications there is hope of averting many catarrhs which tend to become chronic and in their course are liable to produce laryngitis and croup.

The child who is the victim of catarrhal laryngitis should be kept in a well-ventilated large room, of an equable temperature, the air of which is moistened with steam after the method prescribed under Diphtheritic Laryngitis.

Ipecac, in small doses (five drops of the syrup) repeated every half-hour to an hour to the point of nausea, often removes, in mild cases, the harsh, dry respiratory sounds and allows quiet sleep. This is administered on the first night of croup, and makes the child comfortable by mild methods. An added result is, often, a free movement of the bowels and a much improved general condition. The same medication begun in the afternoon of the second day prepares the child for a comfortable and uninterrupted second night. These mild methods of practice commend themselves to the laity. In this connection it is desirable to recommend the triturate tablets of ipecac, of fractions of a grain, prepared by enterprising manufacturing chemists of the day. These have met with great favor in the writer's experience. Triturate tablets so small as one-hundredth of a grain, in young infants, given every ten to thirty minutes for four or more doses, have relieved harsh, dry breathing and given gratifying results.

An opiate (Dover's powder) given at bedtime in dose appropriate to age will often insure an undisturbed sleep to a child whose croupal habit has persisted through other remedies.

In severe cases the bowels should be evacuated with mercurial purge or castor oil. Urgent paroxysms may be met with emetics of ipecac or turpeth mineral (gr. v), repeated, if necessary, in twenty minutes, to insure its action. A hot foot-bath may act as a derivative.

If there is beginning suffocation and fever, with delirium, the wine of antimony (gtt. x) with aconite (gtt. ss) every hour or two hours may be found effective. If the child is frightened and restless, give paregoric in doses suited to the age, to the extent of producing quiet and sleep. If the disease becomes protracted, give one grain of calomel three times a day for two days. Some derive benefit from inhalations of oxygen during the paroxysms of dyspnœa. The following remedies may often be used with advantage,—viz., compresses of ice to the throat, or compresses of hot water by means of a sponge or cloth.

Operative interference is very seldom required. After severe burns from inhalations the most heroic remedies often fail, and operative procedure alone need be considered. (See Intubation and Tracheotomy.)

PSEUDO-MEMBRANOUS LARYNGITIS.

By WILLIAM PERRY NORTHRUP, M.D.

Synonymes.—Croup, Laryngeal diphtheria, Fibrinous laryngitis.

The mucous membrane of the larynx, when inflamed, may have upon its surface a pellicle which is called a pseudo-membrane, composed, for the most part, of fibrin, pus, and necrotic epithelium, and may include necrotic submucous tissue.

A pseudo-membranous laryngitis may arise from trauma: it may arise from the presence of the pathogenic germ or germs of diphtheria: it is believed by many that a similar pseudo-membranous laryngitis may arise from a cause apart from either. This form of inflammation is termed a croupous inflammation.

When of traumatic origin, it is commonly the result of inhalation of steam, hot smoke, irritating vapors, of inspiration of corrosive poisons, etc. These, by destruction of the protecting epithelium of the mucous membrane, allow transudation of serum and extravasation of white blood-cells, with the formation of pseudo-membrane.

In the croupous inflammation which is apt to accompany diphtheria, destruction of epithelium (coagulation necrosis) takes place, and is believed to be due to a specific germ.

The third form is not of traumatic origin, and is not accompanied with pronounced symptoms of the general infection which characterizes diphtheria.

It is with pseudo-membranous laryngitis of the second and third classes that this paper has to do. The writer believes it is not possible in the present state of knowledge to separate purely local croupous laryngitis from laryngeal diphtheria of mild type. It seems advisable, therefore, to consider all cases of pseudo-membranous laryngitis not of traumatic origin as local manifestations of diphtheria, and base the treatment on that diagnosis. For what is to be said concerning the nature of diphtheria as an acute infectious disease, the reader is referred to the subject under its appropriate title. It will be sufficient here to speak of the disease when it has invaded the larynx, either primarily or secondarily. Its etiology must obviously fall under consideration mostly with the general disease.

As frequent reference will be made in this paper to the autopsy records of the New York Foundling Asylum, it may be well at the outset to state that the recorded cases of laryngeal diphtheria amount to one hundred and fifty-one, of which the first eighty-seven cases have been considered by themselves in a separate group. The reason of this separation is that this number represents endemic cases distributed over several years, and therefore more useful to the present purpose. The aggregate number includes in addition the ravages of a fatal epidemic of measles and scarlet fever with diphtheria, with nephritis in most cases and pneumonia in all.

In order that the figures here given may be fully understood, it is necessary to explain the conditions under which they were collected. They represent all the autopsies for a space of six years where diphtheria was found to have involved the larynx, of cases occurring in an institution which has within its walls seven hundred children and has out at nurse in the city and immediate vicinity eleven hundred more. The children are of all ages from birth to five years.

Etiology.—Among predisposing causes may be mentioned *sex*. Though in the asylum the percentage of males was forty-six to fifty in several successive thousands of entries, yet in eighty-seven cases of fatal endemic laryngeal diphtheria fifty were females, and of one hundred and fifty-one cases ninety were females.

Pseudo-membranous laryngitis occurs most often between the ages of one and five years. It prevails in New York in every month of the year. In all localities of the United States, at greater or less intervals, epidemics of diphtheria occur which show a marked tendency to involve the larynx. The disease is moderately communicable between children, but is seldom contracted by nurses and physicians in charge.

In twenty-four cases of diphtheria recently examined with reference to the bacterial origin of the disease, streptococci were found in all but two. This form of bacteria was by far the most abundant of any present in the pseudo-membrane, and the only one which appeared to penetrate the underlying tissues. It was also found in a few cases in the viscera. This streptococcus was demonstrated biologically to be identical with the streptococcus pyogenes and streptococcus erysipelatos and by inoculation into rabbits and pigeons to induce erysipelas, phlegmonous inflammations, abscesses, and localized necrosis.

The crypts of the tonsils were found to be a favorite nesting-place for the streptococcus. In examinations of mouth- and tonsil-scrapings from thirty-one healthy and sick children, not apparently exposed to diphtheria, no streptococci were found except in two cases of scarlet fever, in which diphtheria soon after developed. On the other hand, in examining throat- and tonsil-scrapings from forty children exposed to the disease in a hospital in which it was epidemic, the streptococcus was found in twelve.

The conclusion as stated in the words of the investigator is as follows: "We have seen that all of these observations taken together seem to lead us to so strong a presumption that the streptococcus is the causative factor, in this group of cases at least, of diphtheria, that it practically amounts to a demonstration."[1]

Pathology.—For a discussion of this head the reader is referred to the article on diphtheria. In this paper will be considered only a few points pertaining to the lesion when located in the larynx, together with some complications favored by its location.

The amount of swelling of the mucous membrane and the quantity and consistence of the pseudo-membrane in the larynx may vary very much, and, indeed, may be present in all varieties in the same patient. The concavity of the epiglottis may bear the usual thick, tenacious, yellowish-gray coating, and the false cords be covered with granular, grayish exudate, while the vocal cords and the mucous membrane of the inner circumference of the lower portion of the larynx are only congested and are free from exudate, the trachea at the same time containing membrane of such quality and extent as exists upon the epiglottis. It is common to see the ventricle of the larynx quite filled and obliterated in any case. The exudate in the trachea strips away from the columnar ciliated epithelium without leaving a bleeding surface, and ulceration is more common upon membrane covered with pavement epithelium.

Membranous croup is commonly associated with membranous pharyngitis and tonsillitis, and may be with tracheo-bronchitis. Diphtheria involving only the larynx is not rare, but post-mortem records by no means correctly represent its frequency of occurrence.

In the group of eighty-seven cases of fatal laryngeal diphtheria referred to, the distribution of false membrane was as follows. In nine cases the membrane extended from the tip of the nose to the finest bronchi; in six from the nose to the bifurcation of the trachea; in seventeen from the pharynx to the finest bronchi; in seventeen from the larynx to the finest bronchi; in seventeen from the pharynx to the main bronchi; in seventeen in the larynx and trachea; in three in the pharynx and larynx; and in one in the larynx only. In one case the membrane was well marked from the pharynx to the middle of the trachea. Between this and the bronchi of the fourth division there seemed to be an entire absence of it, and yet in the finest bronchi a distinct membrane could be demonstrated. A tenacious fibrinous cast could be drawn out by the forceps.

Bronchial Diphtheria.—A process which so readily communicates its specific inflammation to the trachea and bronchi, large and small, may be expected many times to involve at last the lungs. In many cases after death tenacious, fibrinous, ramifying processes can be drawn out from the smallest bronchi. This is well demonstrated by making with a long sharp knife a clean section across the base of a lung parallel to and about a centimetre from the base, pressing upon the separated portion, and extruding the fibrinous filaments, which can be grasped by forceps and withdrawn. The diphtheritic inflammation is then seen to have traversed the entire extent of the bronchial mucous membrane.

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[1] Prudden, Etiology of Diphtheria, American Journal of the Medical Sciences, April and May, 1889.

Pneumonia, Broncho-Pneumonia.—Of one hundred and fifty-one fatal cases pneumonia was found in one hundred and four. Its most common location was in the lower posterior portions of both lungs. Recumbent position, gravity, and hypostasis aid in determining the choice of region. To these may be added another element. From the root of the lung, after the division of the main bronchus, one of the largest branches passes to the lower lobe in a line nearly parallel to the posterior margin, giving off at intervals branches to the dependent lung-tissue. Still another branch passes from the root to the lower posterior portion of the upper lobe. It is apparent, from the study of a section with the subject lying on its back, that these two important bronchi are prepared to carry, by gravity, fluid contents from the trachea and main bronchi, for they are so situated as to catch the drainage of the main respiratory tubes. The conditions, too, are favorable,—dorsal decubitus, enfeebled respiratory power, and partial inactivity of the dependent portion of the lungs. The broncho-pneumonia with diphtheria commonly contains a moderate amount of fibrin, but no more than may occur in marked cases apart from it.

Much light has been thrown upon the etiology of pneumonia complicating diphtheria, by the results of some investigations recently made in New York. In an examination, morphologically and by cultures, of seventeen cases of diphtheria complicated by pneumonia, streptococci were found both in the pseudo-membrane and in the lungs in all cases but one. The streptococci from both localities were similar, and when isolated into pure cultures and injected into the trachea of the rabbit induced, uniformly, a lobular and broncho-pneumonia very similar in character to that from which the culture was first obtained. No other species of bacteria was found in these lungs with such frequency and abundance as to justify the belief that it stood in a direct causative relation to the inflammation.

"We arrive finally at the conclusion," says the report of the investigation, "that the acute lobular and broncho-pneumonia which is apt to complicate diphtheria in the upper air-passages in children is, at any rate in the set of cases which we have examined, a form of inspiration pneumonia, induced by the streptococcus diphtheriæ which finds access to the lungs from the foci of diphtheritic inflammation in the air-passages above."[1]

Emphysema.—The interstitial variety occurred eight times in eighty-seven cases, the vesicular variety nine times. The most common location of each is in the anterior portions of the upper lobes. The interstitial may extend to the root in converging lines.

Symptoms and Course.—In a disease so frequently secondary to other maladies, the symptoms must obviously be much obscured and modified by the primary affection. Of the one hundred and fifty-one cases of laryngeal diphtheria, sixty-one complicated measles, ten complicated scarlet fever, and five occurred after a rapid succession of scarlet fever and measles. In fifty-eight cases diphtheria of the pharynx antedated the laryngeal symptoms, and in nearly all other cases, the number not being determined, membrane appeared sooner or later in the pharynx. In eighty-eight cases there was evidence that the membrane made its appearance first in the larynx or at the same time as in the pharynx. *In only one case was it in the larynx only.*

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[1] Prudden and Northrup, Etiology of Pneumonia, American Journal of the Medical Sciences, June, 1889.

It is, then, with diphtheria which either primarily or secondarily invades the larynx, and many times (eighty in one hundred and fifty-one cases) acts as a complication of the exanthemata, that we have to deal. The symptoms of developing and advancing laryngeal complication manifest themselves, in a large number of cases, in the following order,—viz., hoarseness and aphonia, stridulous cough, stridulous inspiration, stridulous expiration, dyspnœa with restlessness, recessions, cyanosis.

Cough.—The first efforts have not the characteristic croupy sound, but rather the sharp explosive sound as of an attempt to remove tenacious mucus from the cords and clear the tone. Later, the explosive element yet remaining, the sound becomes hoarse, then of high-pitched, metallic, or tubular quality, which becomes, in the most aggravated and distressing stages of dyspnœa, dry and whistling.

Stridulous Respiration.—About the time the attention is called to the laryngeal complication, the character of the cough is observed to become more and more metallic, and the respiratory sounds for the first time attract attention. Gradually inspiration becomes stridulous, its sound being low-pitched, hoarse, and the vibrations coarse. Later, as the swelling of the laryngeal walls grows tense, the sounds become high-pitched, prolonged, and metallic. Expirations at first are short and harsh. It is not till inspiratory stridor is well marked that the expiratory takes on similar qualities to the inspiratory, and both become loud, metallic, and at last dry and whistling. At this stage the respirations are quickened, with inspiration and expiration of equal length, both loud, dry, and whistling, interrupted by frequent explosive coughs of similar character.

Restlessness and Dyspnœa.—At the stage of advancement of croup in which the last-mentioned symptoms are conspicuous, there is an equally characteristic nervous irritability, which shows itself in extreme restlessness. This behavior usually marks the beginning of *dyspnœa.* Gradually the respirations grow more frequent, the child sits up, at each inspiration raises his chin a trifle, his nostrils dilate, his face has an anxious expression, his lips are no longer bright red, his color is pale leaden. As dyspnœa increases, and all the inspiratory effort possible on the part of the little patient cannot procure air enough to aerate the blood, he becomes more quiet and succumbs to exhaustion. He lies upon his side, with his knees drawn well up, and his face turned to the wall to avoid being disturbed: his whole attention is centred upon his respiration, and every interference on the part of his attendants is impatiently waved away.

Expiratory dyspnœa from bronchial diphtheria will be mentioned in the further discussion of the course of the disease.

Recessions.—In laryngeal diphtheria with dyspnœa, recessions form a striking symptom. The expansive power of the lungs remains unimpaired, and the muscles of respiration, ordinary and accessory, strive with desperate force to expand the thorax and meet the demands for oxygen. The bony and cartilaginous framework responds, and some air enters, but the glottis is narrowed and respiration hurried. In the great inspiratory effort the soft parts at each end of the thorax yield, and manifest at the supra-sternal notch and supra-clavicular regions inspiratory recessions. At the lower end, likewise, the soft parts yield at the epigastrium; and, lastly, the cartilages are no longer able to withstand the atmospheric pressure, and there are also sternal recessions.

Cyanosis.—Early in the disease, when restlessness and stridulous respiration are the prominent symptoms, the color becomes pale and leaden, with occasional blueness of the lips. On severe coughing, and after sleeping, the duskiness may appear upon the countenance temporarily. As recessions become pronounced, the blood being no longer fully aerated, the lips become blue, the finger-nails dark, and the fingers and face dusky. This may be temporarily relieved, but gradually becomes well seated and remains continuously.

Complications.—*Extension* of the diphtheritic process into the bronchi manifests itself by a rapid rise of temperature, slight increase in rapidity of heart-beats, with increasing tendency to intermittence, and dyspnœa in which the expiration is prolonged and stridulous, inspiration being quick.

Broncho-Pneumonia.—From the main to the minute bronchi is but a short and continuous course. It is at the beginning of this complication that is witnessed the most distressing dyspnœa which the physician is called upon to treat. It is here that the leaden countenance, dull and suffused eyes, livid lips, and livid nails and fingers give their most ghastly picture. Restlessness reaches its highest activity; the child throws himself about, strikes his head heavily against the crib, tears his hair, sits up, throws himself back upon the pillow, asks to be taken on the nurse's shoulder, then put down again wearied, and soon begins again the restless round. As the blood fails more and more of oxygenation and the color of the lips and countenance deepens, the child becomes quiet, lies upon his back, his hands laid upon the pillow, his half-open lids disclosing dusky sclerotics. From this exhausted condition he arouses periodically; again sits up, perhaps takes off one stocking, puts it on again, resting between each change. There is no symptom more remorselessly cruel and no spectacle more piteous to behold than the prolonged secondary dyspnœa of bronchial diphtheria in a child. In the group of eighty-seven fatal cases, eighty-three showed extensive invasion of false membrane; twenty-seven died of bronchial diphtheria, and twenty-nine had pneumonia enough to cause death.

The physical signs are not unlike those of broncho-pneumonia developing after measles. Râles appear early, and give the most accurate information of the advance of the disease, its extent, and its severity. They are coarse or mucous, sibilant, subcrepitant, or crepitant.

The rhythm of respiration is disturbed in pneumonic complications. The child quickly draws its breath, holds it, then with an explosive, grunting sound expires, and without pause again inspires. The pause which in health was after expiration and before inspiration is transferred, and is after inspiration and before expiration. Further irregularities may be observed in the last stages. For a fraction of a minute the child may pause in respiration. In resuming, it may draw a deep breath, and rapidly follow it with short respirations, gradually growing shallower and shallower till respiration again ceases. After momentary suspension the child again draws a deep breath, traversing the circuit from sigh to suspense again and again. Or it may, after the pause, resume breathing by short shallow respirations, followed by suspense. Or a third form may present itself. After a pause the circuit may begin and end with short shallow respirations, having first an ascending scale to the deep respiration, and descending subsequently by similar short, shallow and shallower respirations to the pause. These irregularities in rhythm, so frequently observed in pneumonia following laryngeal diphtheria, have been described by two men whose combined names give to the peculiar phenomena the term "Cheyne-Stokes" respiration.

The disturbance in pulse-rhythm is observed in many severe cases, and is of unfavorable import. It may appear while the prognosis seems still favorable. In one minute the pulse may intermit once, then twice, then drop two beats in a quarter of a minute and not again for two minutes. In dyspnœa the weakness of heart-impulse, combined with strong inspiratory effort, may cause the pulse to fail at the wrist at the instant of beginning inspiration. Though this symptom argues weakness of heart-action, it does not so gloomily point to systemic poisoning as does the former.

Diagnosis.—Progressive unremitting laryngeal stenosis is the characteristic feature of the disease. If to this is added the presence of diphtheritic exudate in the pharynx, the certainty of the diagnosis is assured. If a child having diphtheria of the fauces becomes "croupy," it is not warrantable to attribute such hoarseness to catching cold or to catarrhal inflammation by "sympathy." Muscular spasm not infrequently affects the calibre of the larynx in diphtheritic croup as well as in catarrhal, and many times leads to confusion. Because the croup is remitting it does not follow that it is catarrhal. In many instances early diagnosis as to the variety of inflammation of the larynx is impossible.

Prognosis.—Diphtheria of the larynx, with its complications and sequelæ, is the most fatal disease to which childhood is exposed. Recent methods for relieving the urgent symptoms of stenosis promise to increase somewhat the percentage of recoveries, but the complications are the source of great mortality. Diphtheria beginning in the larynx is unfavorable. It is located at the outset upon respiratory mucous membrane, with a tendency to spread downward by continuity of like tissue into the lungs. In eighty-seven fatal cases, fifty-six began with symptoms indicating that the membrane made its appearance in the larynx before or simultaneously with that in the pharynx. The highest mortality of cases requiring operation attends those under two years of age. Symptoms which indicate that the descent of the diphtheritic process into the bronchi has begun diminish the possible chances of recovery to the minimum. In the above oft-quoted records, the melancholy fact that but one case had diphtheria of the larynx *only*, stands out as a grim reminder of the tendency of the lesion to invade the regions adjoining it above and below.

Treatment.—The remedies for diphtheria are set forth under the appropriate heading, but it may be claimed that the best results obtained by operators for the relief of membranous croup are among those who have made use of bichloride of mercury in rather large doses. One-sixth or one-half or even one grain of the bichloride has been given in divided doses in twenty-four hours and continued at this rate for two to four days. It is not uncommon for operators to adopt this method: to a child of three years give one-fortieth of a grain of bichloride of mercury, in triturate tablets, every hour, followed by a copious draught of water to insure its thorough dilution in the stomach. It is better to dilute it in water sufficient to bathe the surfaces of the pharynx in the act of swallowing, having previously given a draught of water, which should serve to dilute the dose as it reaches the stomach. In any case it must not be forgotten that corrosive sublimate must be in dilute solution in the stomach, to avoid irritation of its mucous membrane. Frequent cleansing of the mouth and the nasal cavity serves to protect the nether cavities from infection borne to them by gravity and insufflation. These are the means recommended to limit the spread of membrane. The medication is usually continued in rather diminishing doses after false membrane has disappeared. Great tolerance for mercury is observed in children with diphtheria. If the bowels are made to move too freely or the stools contain mucus, the most ardent advocates still continue the treatment, adding small doses of opium in the form of paregoric or Dover's powder. At the present time the most popular treatment for diphtheria is, no doubt, mercuric chloride.

Tincture of the chloride of iron is much used. From three to five drops in a teaspoonful of water every hour constitute a very efficient dose for a child three years old. It is best given after rinsing the mouth with water, and serves the double purpose of a beneficial local application and at length of a tonic. Alternating doses of tincture of iron and of corrosive sublimate have been recommended by some.

Potassium chlorate has lost favor, on account of its injurious effect upon the kidneys.

When dyspnœa has made its appearance and is progressing and remains unremitting, more active and decisive procedure is necessary. An emetic may relieve for a time, and should be given. The yellow sulphate of mercury is most reliable and satisfactory, given in five-grain powder, repeating this dose in twenty minutes if emesis is not effected. In an emergency, mustard and water, molasses and sulphur, or warm salt water may be administered to give quick relief. With vigorous effort induced by these medicines, large masses of false membrane may be loosened and expelled. Occasionally the membrane does not return, and the patient is relieved of the croup which it occasioned.

A trained nurse should have charge of these cases, to watch for symptoms under the directions of the physician and to carry out skilfully the details of treatment.

Nourishing food is strongly indicated in this exhausting disease, and there is no food better suited to the needs than milk in as large quantities as can be assimilated.

As a cardiac stimulant, alcohol, in the form of whiskey or brandy well diluted, has met with most favor, and is recommended to be given early and in increasing doses according to the progress of the disease, the condition of the heart, and the age of the patient.

The above is the treatment in vogue at the present time in New York. The disease seems to run a slightly different course in different localities, and remedies applicable to cases in one section have proved of no avail in others.

Benzoate of sodium has been strongly recommended, in doses of eight grains hourly, night and day, to a patient of five years, together with local applications of the same in atomized solution.

Inhalations of oxygen have proved of great value in the late stage of laryngeal stenosis.

To assist by favorable surroundings is desirable, such as filling the air with steam laden with odors of balsam or turpentine or thymol or eucalyptus. Adult patients who are able to express their feelings have approved of steam as a remedy of greatest comfort. It is here recommended to use it in every case, with or without turpentine, and in the following manner. A tent or canopy may be made of blankets. The lower portion of the side opposite to any point of draught may be made to open enough to admit of circulation of air and the entrance of steam. The upper portion, being tightly closed, imprisons the steam which collects in the highest part, and this saturated atmosphere the child breathes, to his great relief. Air for ventilation may with advantage be admitted to the room at any point opposite to the opening of the tent. Steam is best provided by heating a teakettle half filled with water over a grate-fire or gas-stove, and conducting the vapor from the spout to the crib and beneath the tent through a good-sized tin pipe. Such a pipe can easily be procured from a neighboring tinsmith's in the shape of a "leader."

Warm external applications to the neck are to be recommended. An early mercurial purge is given by many with advantage.

One child in ten with well-marked symptoms of laryngeal diphtheria recovers under medical treatment (O'Dwyer).

There comes a time in a large majority of cases when medication must have the assistance of surgical interference. Progressive unremitting dyspnœa which does not yield to medication leaves to the patient but a narrow limit of life without it. To accomplish this relief the practitioner has the choice of intubation or tracheotomy. For the description of each, the reader is referred to their respective titles. It is sufficient here to say that in New York, at least, intubation has largely taken the place of tracheotomy.

INTUBATION.

By WILLIAM PERRY NORTHRUP, M.D.

Definition.—Intubation is the operation for the relief of dyspnœa from laryngeal stenosis, which consists in inserting between the swollen tissues of the larynx a specially-constructed tube, through which the patient breathes. This does not include the temporary expedient of passing a catheter into the trachea, but applies to metallic tubes designed to rest within the larynx for an indefinite time.

History.—The first attempt to introduce a short tube into the larynx was made by Bouchut, of Paris, in 1858. The tube used by him was a hollow metallic cylinder less than an inch long, narrower at one end than at the other,—not unlike a small thimble. This was carried forward on the end of a sound and left wedged in the larynx. Attached to it and brought out at the angle of the mouth was a silken bridle, to secure the tube from passing down into the trachea, and ultimately to remove it. Bouchut published seven cases of intubation in laryngeal diphtheria, in all of which the larynx had tolerated the presence of the tube, and in all the laryngeal dyspnœa had been relieved. These two points were nearly lost sight of in the heated discussion which followed upon the further claims of the author regarding the advantages of "tubage of the glottis" over tracheotomy. The new operation had but few cases, and no recoveries to commend it. The Paris Academy of Medicine appointed a committee, with Trousseau as its chairman, to investigate the merits of the proposed substitute for tracheotomy, and at last accepted their report that it was impracticable. For nearly a quarter of a century there was no further record of any attempt at intubation of the larynx.

In 1880 Dr. Joseph O'Dwyer, of New York, began his experiments in the autopsy-room of the New York Foundling Asylum, quite unaware of the failure of Bouchut and the dictum of the Paris Academy. His first attempt was with a bivalve speculum about an inch long, so adjusted to a handle thrust through it from above as to enter the glottis closed and to spring apart on removal of the staff. This tube relieved the dyspnœa temporarily and was tolerated by the larynx. It was not successful, however, because the swollen mucous membrane gradually protruded between the separated edges of the valves and again obstructed the air-passage.

His next experiments were with solid tubes compressed laterally. These, after numerous modifications as regards length and shape of head or collar, developed into those now made and sold as the O'Dwyer tubes. The aim has been to make an instrument that shall fit the interior of the larynx, extending from just above the false cords to within a half-inch or an inch of the bifurcation of the trachea.

The collar or head, which rests upon the false cords, is irregularly quadrangular, having one angle resting between the arytenoid cartilages and its opposite angle bevelled down or nearly obliterated, the better to allow of closure of the epiglottis over the aperture of the tube. Immediately below the head, the tube is compressed to its smallest lateral diameter, to avoid injurious pressure on the vocal cords. Below this, again, the thickness of the tube-wall is increased by a gradual bulging, which attains its greatest extent midway between the extremities. This bulging below the vocal cords serves to maintain the tube in position during coughing, and increases the weight to be expelled. Towards the lower end the tube diminishes in size, and terminates in a dull edge to enable it to ride harmlessly over opposing surfaces. In this connection it cannot be too strongly urged that each tube should have upon its anterior lower margin a blunt edge so conspicuous as almost to deserve the name of knob,—at least a very thick lip. During each movement of deglutition this portion of the tube rides backward and forward over a limited area of the tracheal mucous membrane, and unless properly guarded will surely remove the superficial epithelium and may excavate the tissues and lay bare the cartilage rings.

Of these tubes there are now in common use six, ranging in size from such as are appropriate for a child of one year or less, up to the age of puberty. The instruments necessary for the operation are—

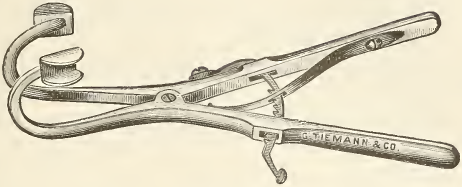
1. The tubes, of various sizes (Fig. 2).
2. Introducing instrument (Fig. 2 *a*).
3. Extracting instrument (Fig. 3).
4. Mouth-gag (Denhard's) (Fig. 1).
5. Gauge (Fig. 4).
6. Braided silk.

Through the edge of the collar of each tube, by an eyelet, is passed a length of braided silk, which is made into a loop or bridle. This serves to remove the tube if it is found to have passed into the œsophagus instead of the larynx, and in case the tube becomes suddenly obstructed by loosened membrane.

The obturator has upon its distal extremity a ball which fills the calibre of the tube and serves both as an obturator and as a blunt end for the entering tube.

The mouth-gag is designed to lock when applied, and remain in place without assistance. It protrudes from the left angle of the mouth, and makes pressure upon the back teeth of the left side. If there are no molar teeth, no gag is necessary. Of the numerous modifications of the original gags, those are to be preferred in which the handles or levers are carried straight back towards the ear.

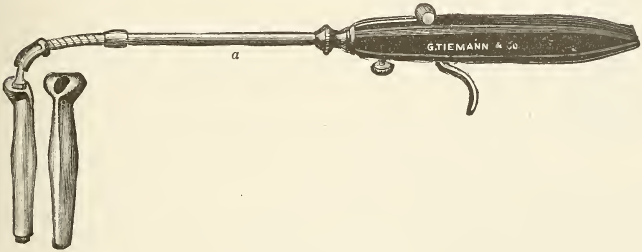
FIG. 1.



Mouth-gag.

The introducing instrument consists of a handle, holding a long staff curved to a sharp right angle at its distal extremity, to which is attached the obturator of a tube of any selected size; also a trigger and sliding gear for detaching it when placed in the larynx. The tube then is held at a right angle to the staff and handle.

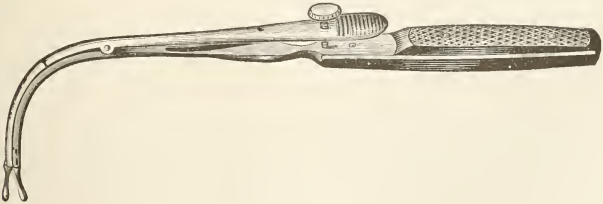
FIG. 2.



Introducing instrument.

The extracting instrument is also curved on a right angle, and carries at its extremity a small forceps with two duck-bill blades, which, by a combination of levers, are made to separate and apply themselves to the interior of the tube with sufficient hold to withdraw it.

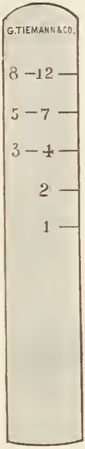
FIG. 3.



Extracting instrument.

The gauge is a measure for determining, from the given age of the child, the size of tube to be used.

FIG. 4.



Gauge.

Braided silk of such size as easily to play through the eyelet of the tube is required for the bridle. Twisted silk may fray out and become jammed in the eyelet.

Directions for Operating.—In attaching the tube to the handle a few trials are made to make sure that the tube holds to the obturator with just sufficient force to carry it into place and yet allow it to be detached by the trigger. Some operators choose a small tube, expecting it to be coughed out after a few hours. Others insert the largest that will enter the larynx, hoping that the larger calibre will allow any mucus and loosened membrane to pass through, and that at the same time the increased bulk and weight will be less easily expelled.

The nurse takes the child upon her lap and quietly wraps it in a light blanket. If the blanket is bulky, it will make large folds below the chin and hinder the operator. The blanket should swathe the child from neck to heels, and be so carefully wound as not to allow the escape of either the hands or the feet. The nurse then grasps the child's elbows, holding them firmly, but in no way interfering with the free expansion of the chest. The legs of the patient are closely clasped by the knees of the nurse. In this way the child is firmly grasped by knees and elbows outside the blanket. The only part movable is the head, and this should be held by an intelligent person standing behind the nurse, preferably by a physician. He grasps the head firmly between his two open hands applied over the child's cheeks and ears. Up to this point the patient usually makes no objection, and it is important that these preparations should be made as here laid down, in order that the subsequent operation may be as exact and speedy as possible.

The position of the child's head, neck, and trunk should be as though he hung from the top of his head, and this should be firmly maintained during the insertion of the tube. It is usual for the nurse to listen to the directions of the physician and then follow them with this result. She takes the child on her lap, winds the blanket tightly around its shoulders and neck, with a huge fold beneath the chin, grasps the child firmly about the chest, settles back in the chair, and catches the child's legs between her knees. The child lies at an angle of forty-five degrees, out of the operator's reach, and the fold of the blanket interferes with the handle of the instruments. While still endeavoring to adjust the gag, the operator will usually find that the child has slipped down from the nurse's shoulder, its feet have escaped and are kicking him in the abdomen and face, and the nurse has nearly squeezed the breath out of the patient's body. *The position of the child should be as though it hung from the top of its head.*

Having placed the child and made sure it is to remain as placed, the operator inserts the mouth-gag between the molar teeth of the left side, the jaws are separated, the levers made fast, and the whole instrument steadied under the hand of the assisting physician. At this point the patient resists more or less energetically, and yet he has rested quietly till within a few moments of the completion of the operation and preserved his strength. The operator usually sits—though some prefer to stand—squarely facing the child, on a firm chair about equal in height to that occupied by the nurse. Holding in his right hand the inserting instrument, having upon its obturator the selected tube, and the silken bridle clear and free, he passes into the child's pharynx the index finger of the left hand and hooks up the epiglottis. Having got this surely up, he sweeps to one side the finger, still holding the epiglottis by its edge. The tube is then carried forward into the pharynx to the end of the left index finger, which is to serve as guide to the entrance of the larynx. At this moment it is desirable to make sure that the instrument is exactly in the median line, and the handle is depressed well upon the child's chest. Having taken these steps, the handle is elevated, the point engages in the larynx and descends to place, the trigger is pressed and the tube disconnected. The tube may be dislocated while removing the obturator, and it is well always to place the finger upon its head and steady it, and after the instrument is entirely disconnected to sink the head of the tube well into the box of the larynx.

The management of the thread is of importance, even after the tube is surely settled into place. It may become entangled, and the tube jerked from its position; on the other hand, loosened false membrane may become wedged in the lower end of the tube, and require its instant removal. In the emergencies which may follow intubation, the operator will be saved some trying moments if he has taken the precaution to secure the thread from accident. It is well to leave the loop long, and fasten it about the ear or to a string encircling the neck. The string should be allowed to remain so long as there is evidence of the presence of tenacious, loose membrane or ropy mucus yet to come away. The author of this paper had been in the habit of removing the thread as soon as the child had thoroughly cleared the tube and respiration had become free. On one occasion, after an easy intubation in a child of four years, the thread had been withdrawn, when suddenly respiration ceased, the countenance became cyanotic, the head dropped forward, and dark blood flowed from the nose. It was not possible to separate the jaws, and tracheotomy seemed necessary. After a gigantic effort on the part of the child, respiration began again, and there came out upon the lip a mass of false membrane shaped like a three-cornered hat, which had been tilted off the tip of the epiglottis and carried before the tube into the trachea. It is true the thread causes great irritation, coughing, and retching, but for a limited time this exertion is of advantage in clearing the air-passages. It is desirable to allow the thread to remain a half-hour or more, and some advocate a longer time.

To remove the thread it is necessary to reintroduce the gag and hold the tube in place while it is withdrawn.

The operation is now complete, and the patient may safely be left from two to four hours, with directions to let him sleep as much as possible and to give nothing to eat but half-teaspoonfuls of milk after a reasonable rest. Cracked ice may be administered without harm, and often affords comfort. It requires a little time for the tube to make peace with the larynx, and for the child to learn to swallow under the new conditions.

In describing the operation step by step, the impression is given that enough time has been consumed to exhaust the patient to a dangerous degree. From the time the child is placed for operation till the tube is in position and the gag is out, scarcely more than thirty seconds need be consumed, and most of this in inserting and adjusting the gag. If the tube is successfully placed at the first attempt, the child is annoyed scarcely more than in depressing the tongue enough to see the lower portion of the pharyngeal walls.

In removing the tube the same position is advisable, though some operators prefer to have the head bent a little forward on the sternum. Here again the epiglottis is hooked up with the left index finger, which again acts as a guide to lead the point of the extracting forceps to the aperture in the head of the tube. In my own experience I have been led to use the palmar surface of the tip of the index finger as the sensitive guide. I pass the point of the finger well down behind the larynx to the beginning of the œsophagus, then lay the finger upon the base of the tongue and partially straighten the finger, at the same time slowly withdrawing it till its tip rests upon the summit of the arytenoid cartilages. As the finger slides over the arytenoids, the epiglottis is carried up. The entrance to the larynx is covered with the pulpy portion of the finger. This position is maintained till the point of the extracting forceps arrives at this most sensitive portion of the guide. Now, if the instrument is held in the median line and the handle raised, the point can be guided with great accuracy to the aperture and the tube removed. New operators, removing the tube for the first time by this method, have reported favorable results.

In certain cases it is desirable to remove the tube to clear it of tenacious mucus, or for other reasons. There is always a possibility that it may be necessary to reinsert it at once and in the face of sudden impending suffocation. For this reason another tube, either of smaller or of similar size, should be threaded and adjusted to the introducing instrument, ready at hand. In this emergency it is worth the knowing that the same tube can be quickly prepared for reinsertion in the following manner. If the calibre is clear, thrust the obturator into the tube and take two turns of thread of any kind around the neck of the tube, gathering the two ends in the right hand as it grasps the handle. In this way the thread holds the tube to the obturator during insertion, and when it is in the larynx unwinds from the shaft and is drawn away. The tube is at such times wet, and threading the needlessly small eyelet of the ordinary tube is difficult and takes valuable time. This simple device stood the author in good stead on one most trying occasion.

After-Treatment.—When the tube has become settled into the larynx and irritation ceased,—in from two to four hours,—a little milk is offered. It may be well to let the child take the cup in its hand and drink as it chooses. Even though it cough after it, and severely, it is able to pass it into the stomach, and by coughing to free the larynx. Again, it may be necessary to try small quantities at a time,—a teaspoonful, while sitting up or lying on the side or back. Semi-solids, such as condensed milk, custards, or corn-starch puddings, may be swallowed without difficulty when milk will irritate. Many times a patient will swallow milk from the nipple of an ordinary nursing-bottle with facility, when he cannot take it otherwise. Elevating the feet to an angle of forty-five degrees, by raising either the bed or the patient, prevents the fluids from entering the tube and trachea. The caprices of childhood know no limit, and often it is the despair of the physician to find anything that the little patient can or will take. One patient would lap her milk after the manner of her pet cat, and in no other way. For five days she took her nourishment with her head forward, lapping from a saucer. In some cases the child refuses or is unable to take sufficient food, and it becomes necessary to resort to rectal injections. Nourishment is so important in diphtheria that it is well to supplement difficult stomach-feeding with infrequent enemata.

It may be desirable to remove the tube temporarily. In many cases the ropy mucus becomes the cause of moderate obstruction and dyspnœa. Before removing the instrument it is possible to aid the patient by a hasty draught of whiskey and water in equal parts. This will often loosen the mucus and stimulate the throat to expel it, to the entire relief of the patient. When this method does not relieve the embarrassment, it is desirable and may be necessary to remove the tube. The larynx after the removal is liable to behave in one of two ways: either the swollen tissues will remain pressed apart and allow air to enter freely for a number of hours, or the relaxed parts, probably the vocal cords, will fall together and give rise to immediate severe dyspnœa. My personal experience leads me to advise that the tube be removed in any case of doubt, and left out as long as possible. It will not be wise for the operator to be beyond easy call for two hours, and the patient is not sure of escaping a second intubation before two days have passed.

The subject of temporary removal includes some discussion of the indications for reinsertion.

Intubation should be performed as soon as air ceases to enter the posterior inferior lobules of the lungs. If on auscultation the ear fails to recognize the characteristic vesicular breathing in these comparatively inactive portions, it can but do harm to postpone relief.

Percentage of Recoveries.—At the present time the statistics collected from published cases place the percentage of recoveries at 26.77 (Waxham, 1888). The number of cases collected was 1027, occurring among many different operators, distributed through several States, and met with in numerous epidemics varying in severity.

Dangers of the Operation.—First of all must be mentioned the accident of pushing before the entering tube loosened plaques of false membrane. It is to meet this danger that the thread is to be allowed to remain attached to the tube for some time after insertions and reinsertions. Secondly, the tube may become obstructed by ropy mucus and require temporary removal. These practically are the dangers which impress themselves upon the mind of the operator.

Intubation has taken its place among the well-established operations, and by many is considered a substitute in full for tracheotomy.

It relieves dyspnœa due to laryngeal stenosis.

There is no objection on the part of parents.

The operation is comparatively free from shock, and free from danger.

No anæsthetic is needed, and no trained assistant.

The subsequent care of the patient does not require skilled attendants.

The inspired air enters the lungs warm and moist.

Intubation does not preclude tracheotomy, and the tube may serve as a guide upon which to cut.

TRACHEOTOMY.

By H. R. WHARTON, M.D.

GENERAL REMARKS UPON THE OPERATION OF TRACHEOTOMY.

Bronchotomy, or the operation of opening the larynx or trachea by an incision through the tissues in the anterior region of the neck, was practised by the earlier surgeons, but its very general adoption in modern times as a legitimate surgical procedure for the relief of tracheal or laryngeal obstruction is largely due to the writings and teachings of Trousseau.

In the present paper I shall confine my remarks to the operation of tracheotomy in reference to diseases and accidents incidental to childhood.

Tracheotomy may be required to relieve the dyspnœa dependent upon membranous laryngitis or diphtheritic laryngitis, growths in the larynx or trachea, growths external to these organs causing pressure upon them, œdema of the mucous membrane of the larynx or trachea, from inflammation, from burns or scalds, or from the inhalation of irritating gases or the swallowing of corrosive liquids. The operation may also be required for the removal of foreign bodies from the larynx or trachea, as well as for the relief of the dyspnœa due to their presence, and it also may be required in cases of fracture or laceration of the larynx or trachea, in cases of spasm of the glottis, and in cases of glossitis, to overcome the mechanical obstruction which prevents the entrance of air into the air-passages.

The prominent symptom arising from these many causes which necessitate the opening of the trachea is a form of obstructive dyspnœa which threatens life, and which is the same in all cases, with possibly the difference in the degree of the obstruction and rapidity of its development. The operation of tracheotomy is in my experience always a most anxious one, for the condition calling for its performance is one which involves a vital function, and, although the surgeon may often be surprised at the ease with which the trachea is exposed and opened in certain cases, yet in others presenting apparently similar conditions he may at each step of the operation be met with difficulties which render it a most formidable surgical procedure. It is, moreover, an operation which is often required in young children, in whom various anatomical conditions obtain, such as shortness of the neck, great vascularity of the parts, the relatively larger size of the isthmus of the thyroid gland, the possible presence of the thymus gland,

and the abundance of adipose tissue, all of which conditions render the trachea difficult to expose and open.

Although some of the above difficulties may be encountered which render the operation an anxious one, yet I am inclined to the opinion of Mr. Marsh, that tracheotomy should be regarded as a delicate operation, which requires coolness and caution in its performance, rather than one which is very difficult or dangerous. I think, therefore, that coolness in the operator is the first requisite, and that, in spite of the alarming symptoms which may be presented, the judicious surgeon will not allow himself to be unduly hurried in his operation, bearing in mind the fact that in cases of obstructive dyspnœa death comes on slowly, except in certain rare instances, that there is more time than at first appears, and that precipitate action at the beginning of the operation may cause much time to be lost before its completion.

The most reliable symptoms of laryngeal or tracheal obstruction are recession of the anterior portion of the chest-wall, and forcible retraction of the epigastrium, the tissues of the suprasternal notch, the supraclavicular spaces, and the intercostal spaces, during inspiration. When these symptoms are marked, we may feel confident that there exists some serious mechanical obstruction to the entrance of air into the chest.

A child suffering from well-marked obstructive dyspnœa has more or less suppression of the voice and presents lividity of the lips and blueness of the finger-tips, and as the dyspnœa increases he becomes restless and cannot breathe in the recumbent posture, sits up in bed and clutches his throat to remove the offending substance, and presents a picture of distress which when it has once been seen cannot well be forgotten. By the change of position the auxiliary muscles of respiration are brought into play, and the restlessness and inability to sleep except at short intervals are explained by the well-known fact that in normal sleep the action of the diaphragm is diminished, but when obstructive dyspnœa is present its action is exaggerated so that sleep is impossible. Labored breathing, which is always observed in cases in which there exists mechanical obstruction to the entrance of air into the lungs, is not to be confounded with frequent breathing, which depends upon diminished air-capacity of the lungs.

At what time tracheotomy should be performed in cases of obstructive dyspnœa is a point upon which there exists some diversity of opinion among medical men. Some recommend the operation as soon as the dyspnœa is well marked, while others postpone surgical interference until the symptoms have become so marked as speedily to threaten life. The operation should not be performed until the dyspnœa is marked and increasing, unless it be due to the presence of a foreign body or a growth in the air-passages or an injury of the larynx or trachea, under which circumstances there is no reason to delay the operation. In cases of membranous laryngitis or inflammatory conditions of the larynx or trachea causing dyspnœa, the surgeon is largely guided as to the time for the performance of the operation by the constitutional condition of the child and by his ability or inability to sleep, for if he can sleep for a few hours at intervals although the symptoms of obstruction are present, I am in favor of postponing the operation, since under such circumstances I have seen very urgent cases recover without tracheotomy; but when the opposite conditions obtain, I think nothing is to be gained by delaying the procedure, for I have never seen such a case recover without operative interference.

The advisability of performing tracheotomy in very late cases is often questioned; but, if an examination of the child shows that he is not dying of cardiac failure, and auscultation of the chest shows that air is entering the lungs and that the membrane has not extended into the bronchial tubes, the urgency of the symptoms presented certainly demands the performance of the operation, for even in the most unpromising cases, where the patients have been apparently moribund at the time of operation, recovery has occasionally followed. The operation usually prolongs life, even if it does not save it, and prevents the patient from dying by a most distressing form of death by strangulation; for in my experience death after tracheotomy from recurrent obstruction is rare, the majority of cases dying of pneumonia or heart-failure or general adynamia.

There is, unfortunately, among people a tendency to regard tracheotomy as a very fatal operation, and to attribute death, if it results after the operation, to the operation itself, and not to the disease which necessitated its performance. For this reason it is often difficult to obtain the consent of parents to have the operation performed upon their children; but this may often be overcome by a candid statement as to what may be accomplished by the procedure.

There is also among the profession too much tendency to look upon the operation as a last resort, and, after it has been performed, to relax the previous local and constitutional treatment of the case; but this is manifestly unwise, for the operation simply fulfils one of the indications,—namely, to remedy the imperfect air-supply,—and it does not interfere with the previous constitutional or local measures which may have been employed.

It therefore may be laid down as a safe rule of practice, that tracheotomy is indicated in all cases of persistent and increasing dyspnœa which is due to mechanical obstruction of the larynx or adjacent parts of the trachea.

SURGICAL ANATOMY OF THE ANTERIOR REGION OF THE NECK.

It is important to bear in mind the arrangement of the anatomical structures of the anterior region of the neck in the operation of tracheotomy, and a brief description of these may not here be out of place.

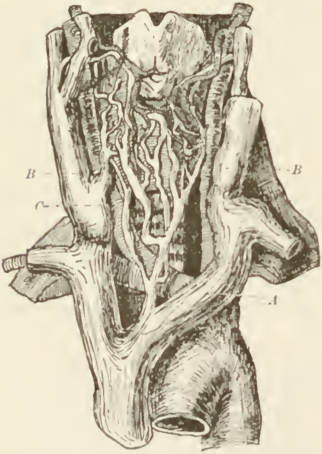
The Fascia of the Neck.—After dividing the skin and superficial fascia, the deep cervical fascia is exposed, consisting of two layers,—the superficial and the deep. The superficial layer is attached to the hyoid bone above by blending with the fascia which attaches the two digastric muscles to that bone, and passes outward and divides to enclose the sterno-cleido-mastoid muscle. Midway between the cricoid cartilage and the sternal notch it again divides into two well-marked fibrous layers, the superficial of which is inserted into the anterior border of the sternum, and the deeper into the posterior border, the interval between them being filled with connective tissue and fat. This layer covers the anterior surface of the sterno-hyoid and sterno-thyroid muscles.

The deeper layer of the cervical fascia beneath these muscles is attached to the lower border of the hyoid bone, enclosing the thyroid isthmus and covering the trachea, and extends into the thorax to join the anterior layer of the pericardium.

Veins of the Neck.—The veins of the neck are most important in their relation to tracheotomy, from the fact that in all forms of pulmonary obstruction they become greatly distended and injuries to them may be followed by very profuse hemorrhage; and they are also most irregular in their distribution.

A large superficial venous branch, the superficial anterior jugular, may be met with in the superficial fascia. The anterior jugular veins, which are very irregular in their course and distribution, are placed superficial to the sterno-hyoid and sterno-thyroid muscles, and are frequently connected by a transverse branch at the lower part of the neck. Usually there is one vein on each side of the median line; one may be larger than the other, or one may cross the median line and empty into its fellow. A plexus of large veins surrounds the thyroid isthmus, opening above into the superior thyroid and below into the inferior thyroid veins. (Fig. 1.)

Fig. 1.



Vessels of the Pretracheal Space: from a child of three years. (After Pilcher.)—*A*, great transverse vein; *B*, *B*, internal jugular veins; *C*, thyroid plexus.

The left innominate vein also occasionally runs above the level of the sternum, and has been exposed during the operation of tracheotomy.

Arteries of the Neck.—The course and distribution of the crico-thyroid, a branch of the superior thyroid, and of the thyroidea ima, an irregular branch from the aortic arch or from the innominate, are of importance, and should be kept in mind by the surgeon during the operation of tracheotomy. The innominate artery occasionally in children rises into the pretracheal space, and this vessel was once exposed by Lücke[1] below the isthmus of the thyroid in performing tracheotomy. There are also occasionally abnormal distributions of the great vessels of the neck which may complicate the operation seriously.

Muscles of the Neck.—The sterno-hyoid and sterno-thyroid muscles are most important landmarks in the operation of tracheotomy. At their upper attachment they are not quite in contact, and as they descend the neck they are farther separated. The space between them which occupies the median line of the neck is an important guide to the operator.

Thyroid Gland.—The isthmus of the thyroid gland, which varies much in size in individual cases and is often largely developed in childhood, is a very important structure in tracheotomy. It usually covers the second and third rings of the trachea, but may extend higher and cover the cricoid cartilage.

Thymus Gland.—The thymus gland sometimes persists and is largely developed in young children, and has been exposed in the operation of opening the trachea below the isthmus of the thyroid gland. In two cases of tracheotomy in which I recently assisted Prof. Ashhurst, in children whose ages were respectively six months and fifteen months, the thymus gland was exposed in the lower portion of the tracheotomy-wound.

Trachea.—The trachea commences at the lower border of the cricoid cartilage and terminates opposite the fourth dorsal vertebra, although its surgical limit is the upper border of the sternum. It is surrounded by loose cellular tissue, is extremely movable, and is most superficial near the cricoid cartilage. It varies in size in different individuals of the same age, being larger in male than in female children. The diameter of the trachea, according to Parker's observations, in children under eight years of age varied from .275 of an inch (6.77 mm.) to .500 of an inch (12.27 mm.).

TRACHEOTOMY IN DIPHTHERITIC OR MEMBRANOUS LARYNGITIS.

In diphtheritic or membranous laryngitis, by far the largest number of cases developing symptoms of obstructive dyspnœa occur among children, and it is in this class of cases that the surgeon is most frequently called upon to perform the operation of tracheotomy.

Indications for the Operation.—The symptom calling for operative interference in diphtheritic or membranous laryngitis is a form of obstructive dyspnœa characterized by suppression of the voice, great difficulty in inspiration, lividity of the lips, depression of the suprasternal and supraclavicular spaces, sinking in of the lower part of the chest, inability to breathe in the recumbent posture, great restlessness, and inability to sleep. When these symptoms are present and increasing, I think that the operation of tracheotomy is urgently indicated; and the only contra-indication to its performance is marked evidence of heart-failure or of occlusion of the bronchial tubes by extension of the membrane.

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[1] Langenbeck's Archiv, vol. iv. p. 589.

Prognosis of Tracheotomy for Diphtheritic or Membranous Laryngitis.—The prognosis in cases of tracheotomy for diphtheritic or membranous laryngitis is naturally more unfavorable than in cases where the operation is performed for simple inflammatory affections of the larynx or for the relief of the symptoms due to the presence of foreign bodies in the air-passages. That this is the case is not remarkable, when we consider the fact that, in addition to the local condition of the larynx or trachea which necessitates the performance of the operation, there exists a most grave constitutional affection which is very fatal in childhood, even in cases where no symptoms of obstructive dyspnœa are developed.

The results following tracheotomy in cases of diphtheritic or membranous laryngitis may best be seen by the examination of large collections of recorded cases. Cohen,[1] in a study of five thousand tracheotomies for croup and diphtheria, found that about one case in four recovered after the operation. In the Hôpital Sainte-Eugénie of Paris,[2] in 2312 tracheotomies there were 509 recoveries, or about 1 in 4.54. At the Hôpital des Enfants Malades, in 2351 tracheotomies there were 614 recoveries, or about 1 in 3.82. Chaym,[3] in 1000 tracheotomies, gives the proportion of recoveries as about 1 in 4. Kronlein[4] reports 504 tracheotomies for diphtheritic croup, with 29.2 per cent. of recoveries. Mastin,[5] in a collection of 863 tracheotomies for diphtheritic croup in the United States, shows that the recoveries were about 26 per cent. Lovett and Munro,[6] in a collection of 21,853 tracheotomies for croup drawn from all sources, show that there were 6135 recoveries and 15,552 deaths, or about 28 per cent. of recoveries.

Individual operators are often able to show a larger proportion of successful results in a limited number of tracheotomies, some being able to show more than fifty per cent. of recoveries; but such statistics are manifestly unreliable, as additional cases would probably diminish the proportion of recoveries very markedly. I have myself recently, in a series of five tracheotomies for diphtheritic laryngitis, had four recoveries, while in six operations preceding this series the result was uniformly fatal. In a series of fifteen tracheotomies recently at the Children's Hospital, there were eight recoveries, a result which even the most hopeful advocate of the operation could not hope to sustain with additional cases.

It will thus be seen, by the comparison of large numbers of collected cases drawn from different sources, that the proportion of recoveries is very similar,—that is, about one recovery in every four cases.

I think it may also be fairly stated that in recent years the results of tracheotomy for diphtheritic laryngitis have been more favorable, depending possibly upon better judgment as to the time of operation, and the greater care which is exercised in the details of after-treatment, as well as upon the improved constitutional treatment of such cases.

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[1] Cohen, Croup in its Relations to Tracheotomy, Philadelphia, 1874.

[2] Mackenzie, Diseases of the Throat and Nose, vol. i. p. 182.

[3] Medical News, 1884, p. 33.

[4] Langenbeck's Archiv, Bd. xxi.

[5] Gaillard's Medical Journal, January, 1880.

[6] American Journal of the Medical Sciences, July, 1887.

Age in the Prognosis.—The recoveries following tracheotomy for diphtheritic or membranous laryngitis in infants and young children are not very numerous, yet there have been enough successful cases to show that age alone is not a contra-indication to the operation in this class of patients. Thus, successful cases are reported at six weeks by Scoutetten, at two months by Steinmeyer,[1] at three months by Annandale,[2] at five months by Croft,[3] at six months by Kisler,[4] and from this age to two years a number of successful cases have been reported. Kronlein,[5] in eighty-five cases of tracheotomy in children under two years of age, reports eleven recoveries. Chaym,[6] in nine hundred and seventy-seven cases of tracheotomy in children two years of age and under, found that only 15.5 per cent. recovered.

Archambault,[7] of the Children's Hospital of Paris, presents some statistics bearing upon the results of tracheotomy at different ages:

Of 976 cases in children from 1 to 3 years of age, 104 recovered.
" 822 " " " " 3 to 4 " " 175 "
" 736 " " " " 4 to 5 " " 174 "
" 497 " " " " 5 to 6 " " 148 "
" 547 " " " over 6 " " 198 "

From these facts it will be seen that age affects the prognosis unfavorably in cases of tracheotomy for diphtheritic or membranous laryngitis; but it must also be borne in mind that the disease for which the operation is performed is itself more fatal in infants and young children.

INSTRUMENTS REQUIRED FOR TRACHEOTOMY.

Under certain circumstances tracheotomy may be performed with very few instruments, but if the surgeon has the choice he will find it convenient to have the following instruments at hand: two small scalpels, one short grooved director, one tenaculum, two aneurism-needles, which may be used as retractors, one pair of artery-forceps, hæmostatic forceps, two pairs of dissecting-forceps, one tenotome, one pair of scissors, one pair of tracheal forceps, one tracheal dilator, tracheotomy-tubes and tapes, flexible catheter, ligatures, sponges, feathers.

The scalpel should be small and narrow in the blade, so that it shall obscure as little as possible the operator's view of the wound.

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[1] Berliner Klinische Wochenschrift, No. 40, 1880.

[2] Edin. Med. Jour., 1862, p. 1121.

[3] London Lancet, November, 1880, p. 849.

[4] Deutsche Med. Wochenschr., No. 45, 1878.

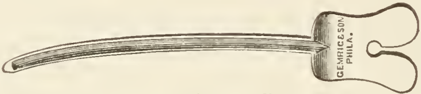
[5] Kronlein, Arch. f. Klin. Chir., Bd. xxi. S. 253.

[6] Medical News, 1884, p. 125.

[7] Archives of Pediatrics, June, 1884, p. 415.

The ordinary grooved director is usually too long to use with satisfaction in the short necks of children, so that I have had made a shorter and slightly broader one, with a bevelled extremity which allows it to be passed with ease between the different layers of the tissues. (Fig. 2.)

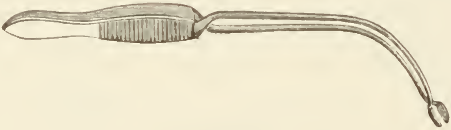
Fig. 2.



Author's tracheotomy-director.

The hæmostatic forceps are of use in case of the division of vessels which bleed profusely, when the operator from the urgency of the case does not think it justifiable to ligature them at the time of their division. They may also be useful in clamping the isthmus of the thyroid gland on either side, where it has to be divided to expose the trachea under similar circumstances. (Fig. 3.)

Fig. 3.

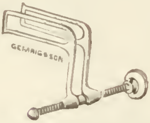


Tracheal forceps.

A sharp-pointed tenotome is the knife I prefer in opening the trachea: its sharp point enables it to be easily thrust into the trachea, and its short cutting surface and narrowness are additional advantages, as they enable the operator to see exactly where he is cutting.

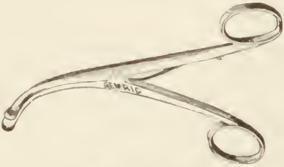
Tracheal dilators, either Golding-Bird's (Fig. 4) or Trousseau's (Fig. 5), are very useful instruments, as they can be slipped into the tracheal incision, and by their use its edges can be held apart until the trachea is cleared of membrane or the foreign body removed, as the case may be, before the tracheotomy-tube is introduced. Golding-Bird's dilator, which is a self-retaining one, is a particularly valuable instrument. Tracheal dilators may be improvised from bent hair-pins or pieces of wire, which may serve the purpose where the ordinary dilators cannot be obtained.

Fig. 4.



Golding-Bird's tracheal dilator.

Fig. 5.



Trousseau's tracheal dilator.

It is well, also, to have at hand a number of pliable feathers, by the introduction of which the trachea and larynx may be cleared of mucus or membrane with little risk of injury to the mucous membrane. The best feathers for this purpose I have found to be the tail-feathers of the turkey.

The Tracheotomy-Tube.—Tracheotomy-tubes of several sizes should be at hand; and it is well to remember that a good tracheotomy-tube is one which inflicts the least possible injury upon the trachea. To insure this, the part of the tube within the trachea should lie exactly in the axis of the trachea, and its free extremity should be capable of as little movement as possible. To accomplish this purpose, the tube should be of the proper shape, and should be large enough to fit the trachea comfortably. Fuller's bivalve canula was formerly generally employed, but it has been superseded by a double quarter-circle canula.

The tracheotomy-tube now in general use is the quarter-circle tube, which is constructed of silver, and consists of two tubes,—an outer one which is attached to a movable collar which fits in a shield to which tapes are attached to secure it in position, and a movable inner tube which closely fits the outer tube. The movable collar, which allows the tracheal portion of the tube to change its position during movements of the trachea and neck, was suggested by M. Roger,[1] and is a modification which has insured both comfort and safety in the wearing of this instrument. The tubes should be of the same calibre throughout, and should not taper towards the lower extremity, as is the case in most of the tubes that are found in the shops. The ordinary tracheotomy-tube usually has a fenestra in the outer tube, but I have never been able to see any advantage in this, as it is generally placed at such a position that it is not continuous with the tracheal canal when the tube is in position, and I think its presence is often a decided disadvantage, as it may cause difficulty in introducing the inner tube by the bulging of the tissues into it. I therefore am decidedly opposed to the use of the fenestrated tube.

The quarter-circle tracheotomy-tube (Fig. 6), made of silver, which is also provided with a fenestrated guide which greatly facilitates its introduction, is the tube which I have found most satisfactory. Mr. Parker[2] recommends an angular tube (Fig. 7) which he has devised and used with success, as he considers that its shape makes it fit the trachea closely and thus prevents erosion of the mucous membrane by its lower extremity, which he considers a danger in the use of the ordinary quarter-circle tube. With the same objection in view, Mr. Durham[3] has devised a very good tracheotomy-tube. Mr. Morrant Baker[4] has devised and used a flexible tracheotomy-tube made of vulcanized red rubber, with good results, and he thinks that by its use the danger of erosion of the tracheal mucous membrane is diminished.

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[1] Archives Générales, 1859, vol. ii. p. 193.

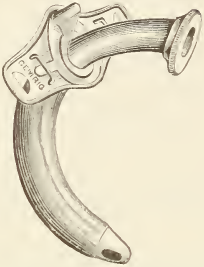
[2] Tracheotomy for Laryngeal Diphtheria, p. 42.

[3] Practitioner, 1869, p. 212.

[4] Med.-Chir. Trans., vol. lx. p. 71.

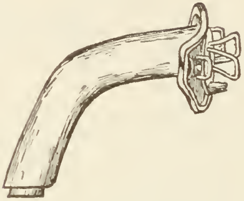
Prof. Little[1] recommends the use of a non-fenestrated tracheotomy-tube constructed of aluminium, which has the advantage of great lightness.

Fig. 6.



Cohen's tracheotomy-tube with fenestrated guide.

Fig. 7.



Parker's angular tracheotomy-tube.

Tracheotomy-tubes constructed of hard rubber have been widely used, but in my experience they are too bulky, and are not adapted for use in recent cases, though they may be employed with advantage in cases where the tube has to be worn for a long time.

The size of the tracheotomy-tube to be employed is a matter of some importance, as the calibre of the trachea varies with the age and with the sex of the patient, for there is no doubt that in female children the trachea is smaller than in males of the same age. The best rule of practice is to introduce a tube which fits the trachea comfortably. I have found that in children under two years of age a No 2 tracheotomy-tube generally fulfils this condition, in children from two to four years of age a No. 3 tube may be employed, while in patients over four years old a No. 4 tube will usually be found satisfactory.

As a substitute for the tracheotomy-tube various forms of tracheal dilators made of wire have been suggested by Watson,[1] Marshall Hall,[2] Bigelow,[3] and Packard.[4] The latter surgeon has constructed such a dilator which is self-retaining and has somewhat the mechanism of the eye-speculum. Experience with the use of these substitutes for the tracheotomy-tube has been very limited, and I am inclined to think they will prove of value only as temporary expedients.

USE OF AN ANÆSTHETIC IN TRACHEOTOMY.

The question of the administration of an anæsthetic in cases of tracheotomy is an important one, and it is one upon which there is much difference of opinion among surgeons. Many operators of large experience are decidedly opposed to the use of an anæsthetic in this operation, on the ground that it is dangerous and unnecessary, while, on the other hand, many surgeons of equally large experience recommend its use, not only as not interfering with the success of the operation, but also as facilitating its performance. There has been, however, in the last few years a growing tendency to discard the use of an anæsthetic in the operation of tracheotomy. Personally I am decidedly opposed to the use of an anæsthetic in the operation of tracheotomy in cases of diphtheritic or membranous laryngitis, for the unfortunate cases which I have seen die during the operation have been those in which an anæsthetic had been used; and I have also seen cases, which were breathing fairly well before its administration, after its use suddenly become so much obstructed that the operation had to be much hurried, and the trachea had to be rapidly opened, often before it was thoroughly exposed, which is a procedure always attended with risk.

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[1] Lancet, August, 1883.

[2] American Journal of the Medical Sciences, October, 1844.

[3] Ibid., July, 1853.

[4] Ibid., July, 1853.

[5] Trans. Penna. State Med. Soc., 1885.

The operation is not a painful one when the dyspnœa is well marked, for after the skin is incised very little pain is experienced in the subsequent steps of the operation. Brown-Séquard has made the observation that an incision of the tissues of the anterior region of the neck causes anæsthesia of the surrounding parts, and hence it is only the first incision which gives rise to pain in the operation of tracheotomy.

A recent paper by Mr. Hewitt[1] will explain the danger of the use of an anæsthetic in cases of obstructive dyspnœa. He says that "in such cases cyanosis is kept at bay not only by compensatory increase in the activity of the nerve-centres which preside over normal respiratory movements, but also by the co-operation of the centres which preside over muscles which take little or no share in ordinary breathing. During ordinary sleep the activity of the diaphragm is lessened, the centres which preside over it enjoying comparative rest; while in obstructive dyspnœa the patient to a great extent depends upon increased action of the diaphragm, so that natural sleep is generally impossible except at short intervals. These vicarious centres will certainly fall victims to an anæsthetic sooner than the automatic or superior centres. The anæsthetic will not, therefore, respect vicarious function, and the muscles will become paralyzed in the usual sequence, and the patients will become more embarrassed in their breathing, or the breathing will cease altogether."

In very late cases it is generally conceded that the use of an anæsthetic is not to be considered. In case an anæsthetic is used, chloroform is probably preferable to ether, as it is not so apt to cause vomiting, and it can be used with safety at night when it may be necessary to bring a light near the wound.

CHOICE OF OPERATION.

There are two points of election in tracheotomy; in one the trachea is opened above the isthmus of the thyroid gland, and in the other below it. These constitute respectively the high and the low operation. In young children the high operation is generally selected, because at this point the trachea is more superficial. In this operation the cricoid cartilage is frequently divided with the upper rings of the trachea.

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[1] London Practitioner, 1887, p. 99.

The low operation, or that below the isthmus of the thyroid gland, is certainly more difficult in its performance, because of the relatively greater depth of the trachea, the large size and number of the veins, and the proximity to the large arterial trunks.

The extreme shortness of the neck in young children sometimes causes trouble in wearing the tube, when the low operation is performed: I call to mind the case of a young child in whom I did the low operation, where it was found impossible to use a tube of the ordinary length, as its lower extremity came in contact with the bifurcation of the trachea, and a shorter tube had to be obtained before the child could wear it with comfort.

Cohen prefers the low operation, and expresses himself decidedly in its favor in cases where the tube is to be worn for some time or where the operation is done for a foreign body impacted in the bronchus.

But as the operation of tracheotomy is, in cases of diphtheritic or membranous laryngitis, done as a temporary measure, and on account of the greater ease and safety of its performance, save in exceptional cases I am strongly in favor of the high operation.

POSITION OF THE PATIENT.

The best position in which to place the patient for the operation of tracheotomy is that which will bring the neck into the greatest prominence; and this can generally most conveniently be obtained by laying the child upon his back upon a firm table, and placing beneath the shoulders a small round cushion; or an empty wine-bottle, or an ordinary roller-pin, wrapped in several towels, will answer the same purpose.

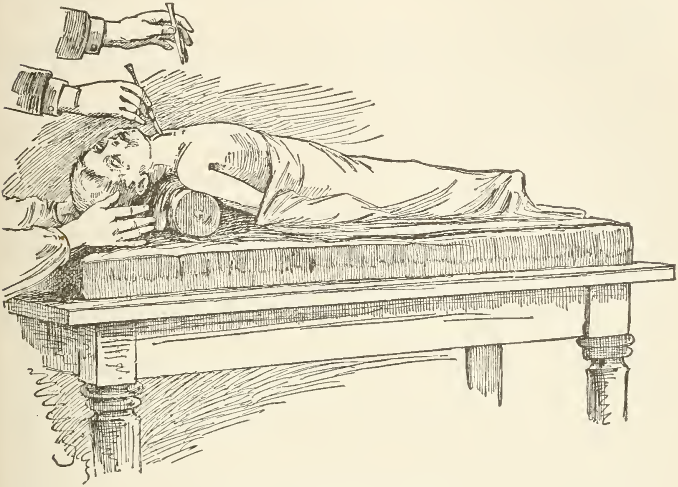
It matters little how the position is obtained, so that the neck is rendered prominent; and it is surprising with how much greater ease the operation will be accomplished if the patient be in a good position.

If an anæsthetic be not used, the arms of the child should be controlled by an assistant or a nurse, which is better than fastening them to the body by a binder pinned around the chest, which may restrict the already embarrassed respiratory movements.

OPERATION.

The child being placed in position, and the head being steadied by an assistant, the operator should take his position either on the right side of the patient or, as I prefer, at the head of the patient, for in this position it is easier to keep the incision exactly in the median line of the neck (Fig. 8). The operator then makes himself familiar with the landmarks of the neck. Locating the cricoid cartilage, he makes an incision in the median line of two or two and one-half inches in length, the position of the cricoid being the middle point. There is no disadvantage in a long incision, and the first incision should divide the skin and expose the superficial fascia. At this point the operator will occasionally see a large vein lying in the superficial fascia,—the superficial anterior jugular vein; this should be displaced, and the fascia should be next divided upon a director.

Fig. 8.



Position of patient for tracheotomy.

The surgeon should endeavor to keep strictly in the median line of the neck, for this is the line of safety, and he should be careful, as the wound increases in depth, not to make his incisions too short, so that it becomes funnel-shaped.

When the deep fascia is exposed it should be picked up and divided upon a director, and any enlarged veins in the line of the wound should be carefully displaced; or, if this is impossible, they should be ligatured on each side and then divided between the ligatures. The operator should now look for the inter-muscular space between the sterno-hyoid and sterno-thyroid muscles: this can generally be found without difficulty, and the muscles can be then separated with the director or the handle of the knife, and the isthmus of the thyroid gland will be exposed. The muscles should now be held aside by retractors placed one on each side. A caution in regard to the use of retractors may not here be out of place: the operator should place them himself and allow the assistant to hold them. I once almost lost a case in which I had the trachea exposed and turned aside to pick up a knife with which to open it, by my assistant replacing one retractor which had slipped, in doing which he dragged the movable trachea to one side, completely shutting off respiration: when I attempted to find the trachea to open it I could simply feel the anterior surface of the vertebræ at the bottom of the wound, and it was only when I lifted the retractor and allowed the trachea to spring back to its normal position that I was able to open it. Mr. Durham[1] mentions a case, and Mr. Marsh also one, in which the trachea and great vessels were held aside with retractors by an assistant until the surgeon had exposed the cervical vertebræ.

The operator should constantly explore the wound with his finger, to locate exactly the position of the trachea and to ascertain the presence of any anomalous arterial distribution.

The isthmus of the thyroid gland being exposed, it is generally found surrounded by a venous plexus, and occupies a position over the first three tracheal rings, or it may extend higher and cover the cricoid cartilage. At this point of the operation he may find that the isthmus of the gland, if large, bulges up and fills the whole wound, and he should endeavor to displace it either upward or downward: this it is often possible to do without difficulty But if it be found firmly fixed, and the trachea cannot be exposed either below or above it, it may be cut through, after being ligatured on each side to prevent hemorrhage. Or a procedure recommended by Bose,[2] which I have employed with advantage in several cases, may be adopted,—namely, a transverse incision across the cricoid cartilage to divide the layer of cervical fascia by which the isthmus is bound down; a director is then passed in, and the isthmus is generally depressed without difficulty.

Having depressed the isthmus of the thyroid upward or downward as the case may be, the trachea, yellowish white in appearance, covered by its fascia, should be exposed, and this fascia should be thoroughly broken up with the director or the handle of the knife so as to bare the trachea. All authorities are agreed as to the importance of thoroughly cleaning the trachea of its fascia before opening it, as by so doing it is easier to incise it and to introduce the tracheotomy-tube. In breaking up this fascia the operator can feel it crepitate under the finger, from the suction of air drawn in with inspiration.

When the surgeon has arrived at this stage of the operation, he may take time to see that the wound is free from hemorrhage, and he may replace the retractors so as to expose as large a portion as possible of the trachea, for, be the case ever so urgent, he now feels assured that he can open the trachea in a moment if the breathing should cease. The trachea should now be fixed with a tenaculum introduced a little to one side of the median line, and an incision should be made into it in the median line from below upward for a distance of one-half to three-quarters of an inch. The use of the tenaculum to fix the trachea has been objected to, but I can see no disadvantage in its use if the trachea is not fixed for too long a time, which arrests respiratory movements, before the opening is made into it. If the trachea be deeply situated, the operator may find it of advantage after fixing it with the tenaculum to lift it slightly from its bed, thereby bringing it more prominently into view and making it more superficial in the wound, thus facilitating its incision.

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[1] Holmes's System of Surgery, vol. ii. pp. 503, 504.

[2] Langenbeck's Archiv, vol. xiv. p. 144.

In opening the trachea I generally employ a sharp-pointed tenotomy-knife. Care should be taken not to introduce the knife so deeply that its point may injure the posterior wall of the trachea or the œsophagus, which accidents have happened by a too deep thrust of the knife. On the other hand, a too superficial incision may divide only the trachea and the mucous membrane, the false membrane, if it be present, not being divided, and the cavity of the trachea therefore not being opened. Now, if the tracheotomy-tube is hurriedly introduced, it may pass between the tracheal wall and the false membrane, and no relief from the dyspnœa will be obtained; I have seen death result from this accident. The importance of a median incision cannot be overestimated; for these wounds are said to heal more promptly, and, if the wound be made to either side of the trachea, the tube does not fit well, and its lower extremity may cause damage to the lateral aspects of the trachea. The moment the trachea is opened, there is generally thrown from the wound with the first expiratory effort mucus or false membrane. This should be wiped away with a sponge and the tracheal dilator should be introduced; the tenaculum should then be removed.

Sudden arrest of respiration sometimes occurs at this time. The entrance of a large body of air, according to Cohen, seems to surprise the lungs, as it were, and there ensues a momentary arrest of respiration, which to one who is not familiar with the circumstance looks like its cessation, and may cause him great alarm at a time when he is about to congratulate himself upon the completion of an anxious operation. This arrest of respiration is generally only momentary, and, if the child's face and chest be slapped with a wet towel, or artificial respiration be employed, the normal respiratory movements will soon be re-established.

Any membrane which appears at the wound should now be removed with a sponge or forceps, and the trachea should be explored both above and below the wound for the presence of false membrane, and if it be found it should be removed by means of forceps, a feather, or a camel's-hair brush. I think that it is owing to the great care which is exercised in this particular, since it has been so urgently insisted upon by Pilcher,[1] Parker,[2] and others, that the results of tracheotomy in diphtheritic cases in the last few years have been so much more encouraging than formerly.

Mouth-suction of the wound has frequently been employed, but as it has been followed by disastrous results to many who have made use of it, and as it is no more efficient in removing the membrane than the forceps, brush, or feather, this procedure cannot be too strongly condemned. Parker has devised a tracheal aspirator for this purpose (Fig. 9), which

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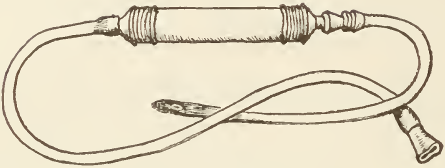
[1] Pilcher, New York Medical Record, 1882, p. 342.

[2] Loc. cit., p. 52.

FIG. 9.



Parker's tracheal aspirator.

consists of a glass or celluloid cylinder three or four inches in length by three-fourths of an inch in diameter, to one extremity of which is attached a flexible tube, and to the other an india-rubber tube with a mouth-piece at the end. The cylinder may be packed with antiseptic cotton, which will act as a filler and prevent any infecting material from reaching the operator's mouth. A flexible catheter of large calibre attached to a syringe may be employed for the same purpose, with good results.

The membrane can be removed with forceps or with a flexible feather, particularly if a little of the soda solution recommended by Mr. Parker be brought in contact for a few minutes with the inner surface of the trachea. The solution to which I refer is as follows:

℞ Sodii carbonatis, ʒi–ʒiiss;
Glycerini, ℥ii;
Aquæ, q. s. ad f℥vi.

A small quantity of carbolic acid may be added to the solution, without in any way affecting its solvent action on the false membrane or mucus. Since my attention was called to this solution, I have frequently used it at the time of operation in clearing the trachea of false membrane, and in the after-treatment of such cases I always use it by means of an atomizer. Of its utility I am firmly convinced.

Mr. Watson Cheyne[1] recommends a solution of the bichloride of mercury, one to five hundred, to be used in touching the raw surfaces after removing the membrane, and he also introduces into the trachea and larynx above the tube strips of lint soaked in a solution of the bichloride, one to two thousand, and washes the wound with a similar solution of a strength of one to five hundred.

The trachea being cleared of membrane, the tracheotomy-tube should be introduced, which can be accomplished without difficulty if the fenestrated guide (Fig. 6) is employed, and secured in position by the attached tapes, which are tied around the neck. The tapes should be firmly tied by several knots, so that there may be no possibility of the child's untying them when not watched by the attendant, as in such an event the tube might become displaced when there was no one competent at hand to replace it.

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[1] British Medical Journal, March, 1887, p. 504.

The immediate results of the operation are, as a rule, most encouraging: the child, who previously exhibited the most distressing symptoms referable to his extreme dyspnœa, now becomes quiet, improves in color, the respiration becomes natural, and it is not unusual to have the patient fall into a quiet sleep before he is removed from the operating-table to his bed.

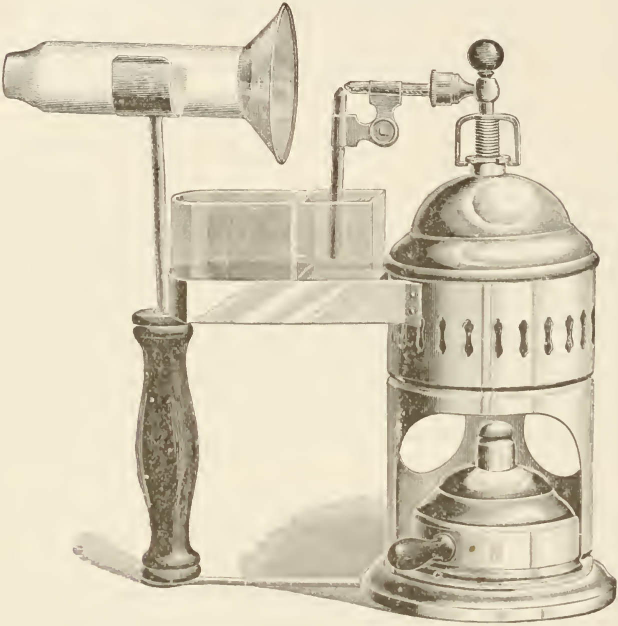
AFTER-TREATMENT OF CASES OF TRACHEOTOMY.

Although the operation of tracheotomy has relieved the patient of the danger of death by suffocation, yet there still exist the same indications for constitutional treatment as were present before the operation: this fact is too often overlooked by physicians, who are apt to relax their efforts in this direction after the operation has been successfully performed. The greatest care is now required also in the local treatment, and I know of no cases in which a successful issue more directly depends upon care and watchfulness in their after-treatment than those in which tracheotomy has been performed for diphtheritic or membranous laryngitis. The patient should also be under the care of an attendant or nurse who is skilled in the management of such cases, who is able to recognize and meet such complications as may arise, and who is familiar with the care that the tracheotomy-tube requires.

After the operation the patient should be placed in a room free from draughts, with a temperature of 70° or 75° F., and the air of the room should be rendered moist and warm by the vapor of steam. In private practice a framework may be fastened over the bed, over which sheets can be stretched, forming a tent. Under this water can be kept boiling in an open vessel, or lime can be slaked. The vapor from the latter Cohen considers one of the most efficient solvents of the false membrane. Or a steam atomizer (Fig. 10) or hand atomizer may be used at frequent intervals, the spray being directed over the opening in the tube. I have found great advantage from the use of Parker's soda solution applied in this manner. The use of steam and the soda solution is especially important if the case is one in which there is little tendency to expectorate false membrane, or if on removing the inner tube it is found clogged with inspissated mucus or membrane. At the Children's Hospital of this city we have a room especially arranged for the treatment of cases after tracheotomy, which is fitted with a steam apparatus by means of which in a few minutes the room can be filled with the vapor of steam and maintained at an even temperature. I think our fair share of successful results at that institution is largely due to this feature of the after-treatment.

If the child coughs and expectorates false membrane after the tracheotomy-tube has been introduced, it may be taken as a good omen, for moist cases, as a rule, are much more favorable than dry cases or those in which there is little or no tendency to expectoration. This clinical observation was, as far as I know, first made by Cohen some years ago, and I have since seen numerous cases which attested its accuracy. In a series of cases of tracheotomy reported by Lovett and Munro,[1] all those in which there was suppression of discharge from the tracheotomy-tube—or, in other words, which could be classed as dry cases—terminated fatally. Our experience at the Children's Hospital has been the same, with one exception. This was in the case of a girl, three years of age, who was admitted to the Children's Hospital in September, 1887, with extreme dyspnœa from diphtheritic laryngitis. In this case I performed tracheotomy, and when the trachea was opened there was no expectoration and it seemed to be a typical dry case, and an unfavorable prognosis was accordingly given. This condition continued for fourteen hours, when, under the persistent use of the steam spray with soda solution, and frequently moistening the trachea through the tube by means of a feather dipped in the soda solution, the child began to expectorate mucus and shreds of membrane, which continued

FIG. 10.



Steam atomizer.

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[1] Loc. cit., p. 167.

for several days. This case finally recovered, the tube being removed on the tenth day.

CARE OF THE TRACHEOTOMY-TUBE.

The nurse having charge of the case should remove the inner tube every hour or half-hour for the first twenty-four hours, and after this time at less frequent intervals, and thoroughly cleanse it with a feather or brush dipped in soda solution, removing any membrane or mucus which may adhere to its inner surface, and should then reintroduce it. The nurse should also be instructed to introduce a soft feather moistened in soda solution into the tube every half-hour or oftener if the case be one in which there is little discharge from the tube, and if there is membrane or mucus loose in the trachea or tube, as evidenced by noisy respiration, this manipulation will facilitate its removal. If a portion of membrane becomes impacted in the tube, its presence will be shown by more or less marked dyspnœa: it can generally be removed by taking out the inner tube, or, if it is not extracted by this means, a feather or the curved tracheal forceps may be employed. If all these means fail and the breathing becomes more embarrassed, the surgeon should remove the tracheotomy-tube, introduce a tracheal dilator, and search for and remove the obstructing membrane, after the removal of which the tube should be reintroduced.

I always caution the nurse not to allow the inner tube to remain out more than a few minutes at a time, for I have seen cases in which it was carelessly allowed to remain out for several hours, when, owing to the tendency of the mucus and membrane to accumulate in the outer tube, it was impossible to reintroduce the inner tube, and the outer tube had to be removed from the wound and cleaned before it could be replaced.

CHANGING THE TRACHEOTOMY-TUBE.

At the end of the second or third day, if the case is doing well, the tracheotomy-tube may be removed and replaced by a fresh one. If a silver tube has been used, black patches may be noticed upon its surface, caused by decomposing discharges or some sloughing spot of the trachea; if such is found to be the case, a tracheal dilator may be introduced and the seat of the trouble exposed, and it can then be treated by the application of a solution of nitrate of silver, glycerin of borax, or a solution of bichloride of mercury in the strength of one to one thousand. At this time the surgeon has also the opportunity of testing the breathing capacity through the larynx, by placing a pad of moistened lint over the wound in the neck. The tube having been thoroughly cleaned or a fresh one obtained, it should be introduced, and, if the fenestrated guide is used, little difficulty is experienced, for the tissues in the region of the wound have become glued together by inflammatory lymph, leaving a sinus leading down to the wound in the trachea when the tube is removed.

The tube need not again be changed for two or three days if there is no special indication for its removal, and it can be left out of the trachea for a longer time at each removal if the child breathes comfortably without it and there is evidence that air passes freely through the larynx.

It is a good plan to allow the nurse or attendant to introduce the tube under the surgeon's direction, so that in the event of its accidental displacement or necessary removal on account of obstruction by membrane she will have learned the way into the trachea and will feel confidence in her ability to replace it. As the case progresses favorably it is well to close the opening in the tube by a cork, which can be kept in place for a short time, and thus test the permeability of the respiratory tract above the wound.

REMOVAL OF THE TRACHEOTOMY-TUBE.

As soon as the child can breathe comfortably with the tube stopped, showing a permeable condition of the larynx, it is advisable to make an attempt to remove the tube permanently. If there is no further indication for its use, its removal is most important, for its presence may set up a tracheitis, which is evidenced by the profuse discharge of glairy mucus, and tracheotomy-tubes which are retained for a long time are in many cases finally removed with the greatest difficulty.

It is impossible to fix a definite time for the removal of the tube in all cases, as the procedure depends upon the condition of the patient and upon the local condition of the trachea and larynx. I have seen the tube permanently removed as early as the third day and as late as the forty-first day, and there are numerous cases recorded in which it has not been possible to remove it for months or even years. In the majority of cases of tracheotomy for diphtheritic or membranous laryngitis I think the tube can be permanently removed from the eighth to the fifteenth day.

After the removal of the tube the wound contracts rapidly, and for a few days the breathing is carried on through both the wound and the larynx, and by the fifth or sixth day after the removal of the tube the wound is generally so far healed that no air passes through it. The superficial wound may then be dressed with a piece of lint spread with ointment and held in position by a strip of adhesive plaster until it is completely healed.

The difficulties in the removal of tracheotomy-tubes in some cases will be considered later on, under Complications after the Operation.

FEEDING OF PATIENTS AFTER TRACHEOTOMY.

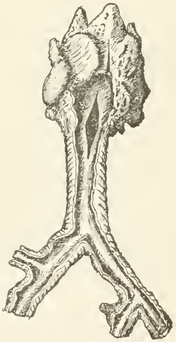
In my experience children wearing tracheotomy-tubes usually take their nourishment well and have no trouble in swallowing fluids, so that they can be given a milk diet or one of semi-solids, or even one of solids if for any reason the latter is considered desirable, without trouble. And it is important to remember that such cases should be given a most nutritious diet. Alcohol in some form should be administered, and, if the appetite fails or the child refuses to take a sufficient quantity of nourishment, rectal feeding or the injection of fluids into the stomach by means of an œsophageal tube should be resorted to.

Sometimes there is regurgitation of fluids through the tube or wound, owing to paralysis of the muscles of the palate: under such circumstances the patient should be given a diet of semi-solids, and if this is regurgitated through the tube or the wound the patient should be fed by means of a soft catheter passed into the stomach, and a syringe, through which sufficient quantities of liquid nourishment may be introduced three or four times during the day; rectal feeding may also be employed at the same time. If it is found necessary to restrict the diet to semi-solids or solids, and thirst is complained of, this may be allayed by the swallowing of pieces of ice, or by the use of enemata of water, care being taken to give small quantities at a time. The surgeon should not give up hope of the final recovery of his patient, even though there be regurgitation of fluids, for I have seen a number of cases in which this complication existed both before and after the removal of the tube, in which by careful feeding recovery finally resulted.

COURSE OF CASES AFTER TRACHEOTOMY.

Many cases after the operation do well for a short time and then terminate fatally from septicæmia, from diphtheritic poisoning, from pneumonia or heart-clot, or from recurrent obstruction due to the extension of the membrane below the seat of operation into the trachea and bronchial tubes. (Fig. 11.) Death from any of the above causes except the last-named is devoid of signs of suffering, and the operation in such cases may be credited with prolonging life and rendering the mode of death much less distressing; but in cases of recurrent obstruction, although life has been prolonged by the operative procedure, euthanasia cannot be claimed for it. Many cases die of heart-clot or pneumonia, and it is a question whether deaths from this complication are more frequent after tracheotomy than in cases of diphtheria in which the operation has not been performed. In diphtheritic cases the incised tissues expose a surface for the absorption of the virus, as is seen by the occasional development on the wound of diphtheritic membrane, and in this way the operation may be said to introduce a small additional element of danger, but it is a comparatively insignificant one, and is not to be compared with the immediately dangerous symptoms for the relief of which the operation was undertaken.

Fig. 11.



Larynx and Trachea from a Patient in the Children's Hospital.—Showing a tubular cast of false membrane of larynx and trachea extending into the bronchial tubes.

Cohen states that croup supervening upon the exanthemata is not, as a rule, amenable to tracheotomy. Lovett and Munro[1] mention seventeen cases in which tracheotomy was performed during the course of some one of the exanthemata: ten of these cases, in which croup complicated measles, gave five recoveries. In the other seven cases, in which croup complicated whooping-cough, mumps, and scarlet fever, the operation failed to save life. I have myself had one successful result out of three tracheotomies performed for croup complicating measles, in a very fatal epidemic of this disease in a Children's Home in this city.

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[1] Loc. cit., p. 169.

COMPLICATIONS AT THE TIME OF THE OPERATION.

Hemorrhage.—The principal complication at the time of operation is hemorrhage, which may be either arterial or venous. It is to be prevented by great care in avoiding the wounding of any vessels of considerable size; if their injury is unavoidable, they should be immediately ligatured, or, if the case is too urgent to admit of delay, they should be secured by hæmostatic forceps, and after the trachea has been opened they can be secured by ligature.

Sudden Arrest of Respiration.—Sudden cessation of the respiratory act during the operation is a most dangerous complication, and it is one which calls for prompt action on the part of the operator. The surgeon's duty under such circumstances is to open the trachea as rapidly as possible, —even through a pool of blood, as described by Mr. Durham,—introduce a tracheal dilator, and make artificial respiration: by such prompt action life can in many cases be saved, and any bleeding vessels can be secured by forceps or ligatures after the trachea is opened.

Mr. Durham says that in those reported cases in which much blood is lost during the operation, and in which this is abandoned before opening the trachea because of the cessation of respiration, death is not the result of hemorrhage, but of failure to complete the operation.

Blood in the trachea after the operation may seriously embarrass the breathing, but if a dilator is introduced it may be cleaned out by the use of a brush or feather.

COMPLICATIONS AFTER THE OPERATION.

Diphtheritic Infection.—Diphtheritic infection of the wound is a complication which is occasionally seen after tracheotomy for diphtheritic laryngitis, and it is one which is not necessarily fatal, although it adds somewhat to the gravity of the case, for I have seen patients recover in whom this condition was well developed. In the treatment of this complication Mr. Parker recommends the local application to the wound of a mixture of one part of hydrochloric acid to two parts of glycerin, which has in his hands been followed by good results. Or the wound may be cleared of membrane by the use of a curette, and the surface may then be swabbed with a solution of bichloride of mercury, one to five hundred.

This condition should not be confounded with sloughing of the wound, with the discharge of thin offensive pus, a condition which is sometimes seen in tracheotomy-wounds in weak and poorly-nourished children.

Inflammatory Œdema of the Neck.—It is only when this condition becomes well marked after tracheotomy that it is a source of danger, for in the majority of cases it exists in the immediate neighborhood of the wound to a limited extent. It is said to be more common in a marked degree in the poor and ill-nourished children seen in hospital practice than in well-to-do-patients in private practice. It may involve the tissues of the neck to such an extent that the tube is lifted out of the tracheal wound by the swelling of the tissues, and a longer one may be required. The treatment of this complication consists in the application of lead-water and laudanum to the inflamed area; if the presence of pus can be detected, it should be evacuated at the earliest opportunity. Stimulants should be administered freely, and tonics are also indicated.

Erysipelas.—Erysipelas, also, may attack the tracheotomy-wound: it commences on the surface, and is generally superficial, but may involve the deeper parts. The treatment of this condition consists in the administration of stimulants, tincture of the chloride of iron, and quinine.

Secondary Hemorrhage.—This is a rare complication after tracheotomy, and it may arise from vessels divided during the operation, or from ulcerative perforation of the trachea from pressure of the lower extremity of a badly-fitting tube causing erosion of some of the great vessels of the neck.

There have recently come under my notice two cases in which death resulted from hemorrhage after the operation of tracheotomy. In one case a profuse consecutive hemorrhage occurred from the tracheotomy-wound some six hours after the operation, and speedily proved fatal. I assisted at the operation in this case, and, although there was some venous hemorrhage at the time, it was thoroughly controlled before the tracheotomy-tube was introduced, and the unfortunate result, in my mind, can be accounted for only by the displacement of one of the several ligatures which had been applied to the injured vessels. The other case was that of an infant six months of age, in whom there was free venous bleeding at the time of operation, which was controlled by ligatures: in this case, on the sixth day a profuse hemorrhage took place from the tracheotomy-wound and tube, and rapidly proved fatal.

M. M. de Heilly[1] showed a specimen removed from a child in whom tracheotomy had been performed in diphtheria, in which the patient died from hemorrhage on the twelfth day after the operation. The hemorrhage in this case arose from an ulceration of the trachea which had extended to the innominate artery and was caused by the end of the tracheotomy-tube. Dr. Hutton[2] reports a similar case in which death occurred on the twelfth day from hemorrhage. Several other cases in which the innominate artery was opened in a similar manner have been reported.

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[1] Brit. Med. Jour., May 12, 1884.

[2] Ibid., 1886, p. 1102.

If the hemorrhage arises from smaller vessels, it is often possible to control it by the application of ligatures or by the use of the galvano-cautery; but hemorrhage from the innominate artery would be so profuse that it would prove fatal before any attempt could be made to control it.

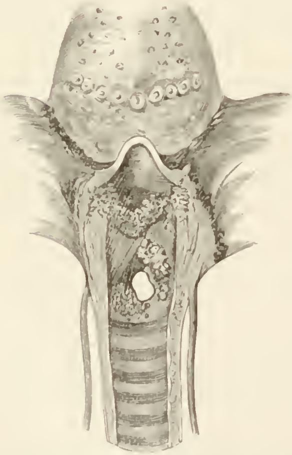
Surgical Emphysema.—Surgical emphysema starting from the region of the wound is occasionally met with after tracheotomy, and it is not uncommon to find it present in a moderate degree, but sometimes the condition is developed to such an extent that the cellular tissue of the neck, face, arms, chest, and abdomen becomes greatly distended with air. A case recently came under my notice in which these parts were all involved, and the crepitation of the air at the ends of the fingers could be distinctly felt; in this case, also, there was great recurrent dyspnœa, which was probably due to mediastinal emphysema.

The presence of air in the tissues is explained by the fact that there is, during the violent inspiratory efforts in obstruction of the larynx, more or less of a vacuum produced in the chest, and the air is sucked into the cellular tissue of the neck and diffused throughout the tissues generally. It is said to be more common after tracheotomies in which the opening into the trachea is not in the median line and does not correspond with the wound in the soft parts in front of the trachea.

Dr. Champneys[1] reported twenty-eight cases in which autopsies had been made after tracheotomy had been performed for diphtheritic laryngitis: in sixteen cases emphysema of the mediastinum was present. The condition was also found in patients dying from diphtheria in whom tracheotomy had not been performed.

Emphysema when developed to a moderate extent seems to do no harm, for the air is usually quickly absorbed; but when it becomes general, and the mediastinum is also involved, dyspnœa is apt to occur, and the prognosis is extremely grave.

FIG. 12.



Granulations in the trachea about the tracheotomy-wound. (After Parker.)

Granulations about the Tracheal Wound.—In certain cases there seems to be a peculiarly hypersensitive condition of the mucous membrane of the trachea, and the presence of a tracheotomy-tube (even a well-fitting one) will be followed by the occurrence of exuberant granulations. (Fig. 12.) These granulations are more commonly seen in cases where tubes have been worn for a long time, and often are a cause of difficulty in their permanent removal. The presence of this complication may be suspected if the child coughs up blood-stained secretion after the tube has been changed. Removal of the tube and inspection of the wound will often disclose the presence of granulations attached to the edges of the tracheal wound or growing from the trachea in the region of the wound.

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[1] London Lancet, August, 1883, p. 1044.

The treatment of this condition consists in the application to the granulations of a thirty-grain solution of nitrate of silver, or touching them with the solid stick of nitrate of silver, which may be fused upon a silver probe bent to a suitable shape.

Ulceration of the Trachea.—This complication may arise from an improperly-shaped or badly-fitting tracheotomy-tube, and its presence may be suspected when the tube, if it is a silver one, becomes blackened and there is fetor of the breath and expectoration; purulent and blood-stained discharge may also occur. I do not think that with the improved tubes now in use this complication is so apt to occur as formerly. The treatment of this condition consists in the application to the ulcerated portion of the trachea of a ten-grain solution of nitrate of silver, and the badly-fitting tube should be replaced by a properly-fitting one.

Difficulties in the Permanent Removal of the Tracheotomy-Tube.—Although in the great majority of cases the tracheotomy-tube can be permanently dispensed with in from eight to fifteen days, yet there are occasionally met with cases in which this cannot be accomplished for months or even years, and a few cases have been recorded in which its final removal was never satisfactorily accomplished. In some of these cases the difficulty is due to mechanical causes, such as the growth of granulations in the trachea near the wound or in the larynx, inflammatory hypertrophy of the vocal cords, adhesions between the cords, paralysis of the posterior crico-arytenoid muscles, spasm of the glottis, or stenosis of the trachea at the seat of operation. Dr. Emil Köhl,[1] in an exhaustive article upon this subject, mentions also, as a cause of delay or difficulty in removing the tracheotomy-tube, prolonged diphtheria, re-formation of the diphtheritic membrane, changes in shape of the trachea or larynx from the operation or from the wearing of the canula, and relaxation of the anterior wall of the trachea.

Where the difficulty in the permanent removal of the tube is due to the presence of granulations in the trachea or larynx, their removal by some of the methods before mentioned will generally enable the patient to dispense with the use of the tube.

When stenosis of the trachea or larynx exists and prevents the permanent removal of the tube, the parts may be gradually dilated by the use of bougies; or, what is better, an intubation-tube (O'Dwyer's) may be introduced on the removal of the tracheotomy-tube, and the wound in the neck can be plugged with a nipple attached to a shield (Fig. 13), to keep the wound from healing until it is certain that there will be no further necessity for the reintroduction of the tracheotomy-tube. The intubation-tube may be worn for some weeks and then removed, and, if the breathing is satisfactorily carried on with the wound in the neck plugged for several weeks, this may then be allowed to heal. I have now under my care a boy who wore a tracheotomy-tube for four years, in whom after its removal an intubation-tube was introduced, which he is now wearing with comfort, and with a fair prospect of being able to dispense with it in a short time.

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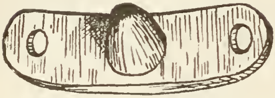
[1] Langenbeck's Archiv, Bd. xxxv., 1887, pp. 75, 403.

Fig. 13.



Plug with shield to keep tracheotomy-wound from healing. (After Parker.)

I have also seen difficulty in some cases, especially in young children, in whom the trachea is very flexible, in removing the tube, from the fact that the wound in the soft parts in healing had become attached to the tracheal wound, and in inspiration assumed a valvular form, allowing little air to enter. If the larynx is not clear or there is irregular action of the laryngeal muscles under these circumstances, dyspnœa soon becomes marked, and the tube has to be reintroduced. This can be overcome by removing the tube from time to time and trying to induce the child to learn again to breathe through the larynx, or by applying an intubation-tube for a time, observing the same precautions with regard to the prevention of the healing of the tracheal wound until breathing can be satisfactorily accomplished through the larynx.

Mr. Thomas Smith[1] has shown that tracheotomy is apt to cause undue irritability and disorderly action of the muscles of the glottis, so as to interrupt their usual rhythm. Cohen[2] says that the explanation of these phenomena resides in the fact that the laryngeal muscles have lost their habit of contracting harmoniously with the needs of respiration, the patients being somewhat in the condition of those with paralysis of the vocal cords. Some cases can breathe comfortably without the tube except during sleep, and in explanation of these Mr. Thomas Smith suggests that the influence of the will may be necessary to regulate and secure due action of these muscles, the perfection of whose movements has been impaired, and that on this account inspiration through the larynx during sleep is impossible.

In other cases mental agitation plays an important part in preventing the removal of the tube; for a child who can breathe comfortably through the larynx when the tube is plugged, or when it has been removed and the tracheal wound has been closed with the nipple-shaped plug (Fig. 13), will on the removal of either of these exhibit great mental agitation and develop such alarming symptoms of dyspnœa that the reintroduction of the tube becomes necessary. Indeed, it is remarkable to observe how even a very young child will depend upon the presence of the tube for breathing, and how he will resist its removal, and often will get into such a rage if it is removed that the rhythmical respiratory actions are so much embarrassed that it becomes necessary to replace it. Cases have been recorded where children would breathe comfortably only, even after the wound had healed, by having the tracheotomy-tube tied around the neck. Stevenson[1] makes the observation that fright at the removal of the tube in children produces a nervous excitable condition, the irregular inspiration and sobbing seeming to induce spasm of the glottis.

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[1] Med.-Chir. Trans vol. xlviii. p. 227.

[2] Loc. cit., p. 53.

If there is no mechanical difficulty present to prevent the permanent removal of the tube, it will be found that by gaining the confidence of the child, and by patience and perseverance in withdrawing the tube at intervals of gradually increasing length, its removal can in most cases be finally accomplished.

Post-tracheotomic Vegetations.—In addition to the vegetations or granulations which occur in the region of the trachea about the wound before the tube has been finally removed, there have been described under the above title growths which occur in rare cases in the trachea after the wound has cicatrized. These growths are more apt to occur in male children, and appear from fifteen days to a month after the wound has healed.

The symptoms of this affection are embarrassed respiration with progressive dyspnœa. The first case of this nature was reported by Gigon,[2] and since that time some fourteen cases have been collected by Ross.[3] Recently Dr. Denger[4] reported a case which died two weeks after the wound had healed, in which an autopsy revealed a tumor of granulation-tissue in the trachea at the seat of the tracheotomy-wound.

The treatment of these growths consists in opening the trachea, removing them with scissors or a knife, and cauterizing their bases and introducing a tracheotomy-tube. If they show no tendency to recur after a short time, the tube may be withdrawn and the wound allowed to heal.

TRACHEOTOMY WITHOUT TUBES.

A number of surgeons, recognizing the amount of attention which patients require while wearing tracheotomy-tubes, and possibly overestimating the dangers in their use, and the difficulty which sometimes is experienced in finally removing them, have recommended and practised the operation of tracheotomy without the use of a tube. Dr. Martin[5] has reported several successful cases of tracheotomy in which he dispensed with the use of a tracheotomy-tube, the edges of the tracheal wound being stitched to the skin. Other surgeons have removed small portions of the trachea on each side of the incision when no tube was used.

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[1] Guy's Hospital Reports, 1875.

[2] Mackenzie, Diseases of the Throat and Nose, vol. i. p. 526.

[3] Edinburgh Med. Jour., 1883, p. 789.

[4] New York Medical Record, 1882, p. 583.

[5] Trans. Amer. Med. Association, vol. xxix. p. 219.

The number of cases in which the use of the tracheotomy-tube has been entirely dispensed with has been so small that we cannot as yet fairly judge of the value of the procedure; and, moreover, there is very little danger in the use of the tracheotomy-tubes which are now generally employed, if the precaution be taken to see that they fit the trachea well. The objection that more care is required in the after-treatment of the case while wearing a tube is not a valid one, as it seems to me that an equal amount of attention would be necessary after the operation, whether a tracheotomy-tube were used or dispensed with. I therefore am decidedly of the opinion that the use of a well-fitting tube is a most important factor in the successful issue of a case of tracheotomy, and as such would most strongly recommend its employment.

THERMO-CAUTERY IN TRACHEOTOMY.

The dread of hemorrhage has led certain surgeons to substitute the thermo-cautery for the knife in the operation of tracheotomy. In 1870 Amussat[1] first employed galvano-cautery in tracheotomy, and this method has also been employed by Verneuil,[2] Krishaber,[3] and others.

RAPID TRACHEOTOMY.

Some operators, on the other hand, have so slight a dread of hemorrhage in the operation that they recommend a rapid tracheotomy by a single cut. De Saint-Germain[4] claims to have performed a number of such tracheotomies without a single grave accident due to the operation.

Mr. Durham[5] has recommended a rapid tracheotomy which he performs in the following manner. The surgeon stands upon the right side of the patient, and places his forefinger on the left side of the trachea and his thumb on the other, so as to include between them the spot at which the trachea is to be opened: firm pressure is made, and the trachea can be felt between the thumb and finger; the safety of the great vessels is insured, as they are outside of the line of incision. By a succession of careful incisions the operator cuts down on the trachea, and when it is exposed he may open it directly, or fix it with a tenaculum before opening it. Mr. Durham claims to have operated upon a number of cases by this method, without any untoward results.

None of the above methods of performing tracheotomy have been very generally employed, and I fail to see their superiority over the slower and safer method of dissecting carefully down to the trachea. I therefore do not think they will supersede the latter operation, which has the advantage of enabling the operator to recognize and avoid structures the wounding of which would be dangerous.

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[1] Bull. de Thérapeutique, 1872, p. 472.

[2] Bull. de l'Acad. Méd., 1872, p. 299.

[3] Mém. de la Société de Chirurgie, 1874.

[4] Cohen, loc. cit., p. 715.

[5] Practitioner, 1869, p. 227.

CONDITION OF PATIENTS AFTER RECOVERY FROM TRACHEOTOMY FOR DIPHTHERITIC LARYNGITIS.

The condition of patients after recovery from tracheotomy for diphtheritic or membranous laryngitis is a matter of some interest. As far as my personal observation goes, the voice seems to be unimpaired, and these patients do not seem to be more liable to laryngeal affections than those in whom recovery has followed without operative interference. The rare occurrence of post-tracheotomic vegetations has been previously mentioned. Drs. Lovett and Munro (*loc. cit.*) have made some very valuable observations upon this subject. They report that in fifty-six cases where tracheotomy had been performed more than a year previously, which they investigated with reference to the effect of the operation upon the voice and general health of the patients, fifty-three were in good health, and none of them had had a second attack sufficient to call for surgical aid. The voice was clear in all but four cases, six patients were liable to sore throat, and three were not in good health,—one having phthisis, but without any laryngeal symptoms, one a hoarse and croupy voice, and the third being a delicate boy who was constantly ill.

TRACHEOTOMY FOR FOREIGN BODIES IN THE TRACHEA, LARYNX, OR BRONCHUS.

Foreign bodies may gain access to the air-passages either by the mouth or by perforation of the wall of the air-tube: the former class is by far the more numerous, and generally consists of substances which being held in the mouth are suddenly drawn into the air-passages by an inspiratory effort. The natural tendency which exists in children to place all conveniently-sized articles which come into their possession into their mouths causes the variety of bodies which find their way into the air-passages of this class of patients to be very great. An examination of the works of Gross[1] or Poulet[2] will give some idea of the great variety of these objects; probably the most common of the offending bodies met with are pins, needles, beads, peas, buttons, beans, pebbles, and grains of Indian corn. The latter object, according to Weist,[3] is the most common foreign body met with in American cases.

When a body gains access to the air-passages, its presence gives rise to persistent cough and violent expiratory efforts, which if the body be of suitable shape may cause it to be expelled. In other cases the body may become impacted in the larynx, and, if its size be sufficient to cut off the air-supply, death from suffocation will usually take place before surgical aid can be procured. If, on the other hand, the body is small, it may pass through the larynx and enter the trachea or one of the primary bronchial tubes and become impacted or remain movable. The tendency of the foreign body to pass into the right bronchus rather than into the left is probably due to the fact that the septum at the bifurcation of the trachea is somewhat to the left of the median line, rendering the right bronchus larger than its fellow. If it is movable, it will change its position with the movements of the current of air in respiration, and it generally causes the greatest difficulty in expiration; whereas if it be impacted or fixed, the difficulty will be most marked in inspiration.

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[1] Gross, Foreign Bodies in the Air-Passages, 1854.

[2] Poulet, Foreign Bodies, p. 22.

[3] Trans. Amer. Medical Association, vol. i., 1883.

Prognosis in Cases of Foreign Bodies in the Air-Passages.—The prognosis of cases in which foreign bodies are present in the air-passages is always grave, for, aside from the dyspnœa and inflammatory accidents consequent upon their presence, the bodies may change their position and cause death from sudden occlusion of the air-current; but this accident, according to Cohen,[1] is not so frequent as might be supposed, if the bodies have occupied the air-passages for some time.

Weist,[2] in an analysis of one thousand cases of foreign bodies in the air-passages, gives 76.92 per cent. of recoveries in those not operated upon, and 72.48 per cent. of recoveries in cases subjected to tracheotomy. It would thus seem that the proportion of recoveries was larger in those not operated upon, or in which spontaneous expulsion took place; but, as justly remarked by Cohen in commenting upon these figures, it is to be presumed that the severest or most urgent cases have been treated by tracheotomy and the milder ones have been treated expectantly, and that the recoveries would have been more numerous if there had been a greater proportion of operations.

On the other hand, the results in Mr. Durham's[3] collection of cases were much more favorable when tracheotomy was performed than when the cases were treated expectantly. In his collection of 636 cases of foreign bodies in the air-passages, in 338 cases in which tracheotomy or other operative procedure was adopted the mortality was 23.08 per cent., while in 298 cases treated expectantly the mortality was 40.94 per cent.

Ashhurst[4] states that, if the cases treated by expectancy in both Weist's and Durham's collection be combined, we find a total of 897 cases treated without operation, with 261 deaths, or a mortality of 29.09 per cent., nearly 6 per cent. more than when operative measures were adopted.

Holmes[5] gives the result of 212 cases of tracheotomy for foreign bodies in the air-passages, which are as follows: 157 recoveries, or 74.6 per cent., and 55 deaths, or 25.4 per cent.

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[1] International Encyclopædia of Surgery, vol. v. p. 672.

[2] Trans. Amer. Surgical Association, 1882.

[3] Holmes, System of Surgery, vol. i. p. 765.

[4] Ashhurst, Principles and Practice of Surgery, p. 368.

[5] Loc. cit., p. 770.

Treatment in Cases of Foreign Bodies in the Air-Passages.—In view of the dangers consequent upon the presence of a foreign body in the air-passages, at the present time the weight of surgical opinion is decidedly in favor of an attempt to remove it through an incision into the trachea, if it has passed below the vocal cords and cannot be removed by means of laryngeal forceps introduced through the mouth. When the foreign body is lodged in the larynx, its location by laryngoscopic examination and its removal by laryngeal forceps in skilful hands is a safe and efficient procedure; but it will be found that most patients, and especially children, require a certain amount of training before the ordinary laryngoscopic manipulations can be satisfactorily accomplished.

The treatment of foreign bodies in the air-passages by the administration of emetics, while not entirely free from danger, has proved of little service in their removal. Durham mentions fifty-three cases in which this method was employed, in forty-six of which it proved useless. Inversion of the body is now generally regarded as an unsafe method of treatment, from the risk of the foreign body becoming impacted in the chink of the glottis, unless there has been previously made an opening into the trachea, or unless the surgeon is prepared to perform an immediate tracheotomy if dangerous symptoms arise.

The presence of a foreign body in the air-passages, in addition to exciting a persistent cough, is soon followed by symptoms of dyspnœa more or less marked according to the size and situation of the body. If the symptoms become urgent, the patient is best relieved by tracheotomy, and the choice of operation rests between the high and the low operation.

In view of the rapidity and safety with which the high operation can be performed, it is decidedly to be preferred if the foreign body is in the trachea or the larynx, as in the latter case the larynx can be more conveniently explored from the wound of the high operation than from that of the low one. On the other hand, if there is reason to believe that the body is impacted in one of the primary bronchial tubes, the low operation should be selected, as it gives the operator a better opportunity of reaching and removing the offending substance.

If the dyspnœa be urgent, from the presence of a foreign body in the air-passages, the same objection exists to the administration of an anæsthetic as in cases of croup: if employed at all, its use should be confined to cases in which the dyspnœa is not marked.

The steps of the operation for the removal of a foreign body from the air-passages by tracheotomy are similar to those when it is undertaken for the relief of obstructive dyspnœa due to croup. The operation may have to be more rapidly performed, by reason of the urgency of the symptoms presented, or the symptoms may become more urgent during the operation, or the respiration may cease from the foreign body changing its position.

The greatest care should be taken to avoid wounding any considerable vessels, the bleeding from which would delay the operation, and, if there is time, the trachea should be well exposed before it is opened. The trachea being exposed, it should be fixed for a moment with a tenaculum, and an incision, strictly in the median line, should then be made into it from below upward, dividing three or four rings of the trachea. The tracheal wound should be longer than that which is made to introduce the tube in cases of croup, so as to facilitate the expulsion or removal of the foreign body. As soon as the trachea has been incised for a sufficient distance, a dilator should be introduced, and the edges of the tracheal wound should then be held apart, and, if the foreign body be movable and of a size to pass through the wound, it is usually expelled with the first forcible expiration. If, on the other hand, the body is fixed or impacted in the larynx, the trachea, or a bronchus, its position can usually be located by introducing a flexible probe or catheter through the wound and exploring the canal, and when it is found it can generally be removed by the use of tracheal forceps.

The foreign body having been expelled through the tracheal wound or removed by forceps, and all bleeding having been controlled, the question arises whether it is advisable to attempt to close the wound in the neck,—a question which the surgeon has to decide in each individual case. If the foreign body has been in the trachea for only a short time and has been removed without difficulty, the introduction of a tracheotomy-tube is unnecessary, and the surgeon may close the wound by the introduction of deep sutures, or by two sets of sutures,—one deep and the other superficial,—and attempt to get union in the line of the wound. It is not often that this can be obtained; so that some operators satisfy themselves with introducing a few sutures at each extremity of the wound and leave the central portion open, and others introduce no sutures, leaving the wound open to heal by granulation. Immediate suture has recently been advocated by Sir W. MacCormac and Mr. Morris in wounds of the trachea; and this procedure might with advantage be employed in these cases. The wound should be covered with a few layers of gauze kept moist with soda solution or weak carbolized solution, as long as air continues to pass through the tracheotomy-wound.

If the body has been in the air-passages for some time and has set up inflammation of the mucous membrane of the larynx or trachea, it is not well to attempt to close the wound; in such a case it is better to introduce a tracheotomy-tube and allow it to remain for a few days, until the inflammation has subsided. The patient should be placed in a room with a temperature of about 70° F., and care should be taken to keep the tube clear of discharge, which is often profuse if there has been much tracheal inflammation: the use of the steam spray of soda solution by inhalation will be found most efficient for this purpose. If the case does well and discharge from the tracheal wound diminishes, after a few days the tube can be removed, and the wound may then be allowed to heal by granulation.

The importance of introducing a tracheotomy-tube in cases where the foreign body has set up much tracheal irritation was well shown in a case of tracheotomy in which I assisted Prof. Ashhurst recently. The patient was a child under two years of age, who three days before his admission to the hospital had got a portion of a grain of Indian corn into his trachea, which was followed by dyspnœa, which steadily increased and was most urgent when he was operated upon. In this case, when the trachea was opened a portion of a grain of corn was removed from the trachea near the wound; but there was so much inflammatory softening of the trachea and swelling of the mucous membrane that in inspiration the lower portion of the tracheal wound was drawn downward and the trachea was flattened, so that little air could enter, and it was only when the wound was kept patulous by a retractor that the child could breathe. As soon as a tracheotomy-tube was introduced the breathing was satisfactorily carried on. The child ultimately recovered.

If upon exploring the wound it is found that the body cannot be located or removed, the wound should be kept open for some time with retractors or by a tracheotomy-tube, and it should be explored at intervals to ascertain if the foreign body can be located. A case presenting such conditions should be carefully watched, so that the tracheotomy-tube might be removed and the wound dilated if the body became loose and, failing to pass through it, became impacted in it or became fixed against its lower extremity.

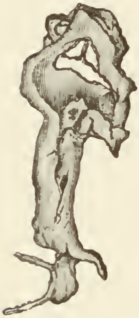
The amount of relief which is given to the dyspnœa by opening the trachea, even in cases in which the body is impacted below the seat of the tracheal wound and cannot be removed, is remarkable, and shows that in such cases there is often a reflex laryngeal spasm. A few years ago I had under my care a child who presented urgent symptoms of dyspnœa which had come on after getting a steel pin in her air-passages on the previous day. Tracheotomy relieved her symptoms, although the pin could not be found at the time of operation; it was afterwards located in the left bronchus, and was finally expelled several months afterwards, the child completely recovering.

Occasionally a sharp-pointed body, as a pin or a needle, may become impacted in the trachea or larynx, and its point, gradually working its way through the walls of these organs, may be felt under the tissues which cover them. These cases, if the body cannot be removed by the use of the laryngoscope and forceps, are best treated by cutting down upon the body from without, and, enlarging the wound in the trachea or larynx, withdrawing it. At the Children's Hospital, such a case came under my care, in which a little girl had got a pin into her larynx, and when she presented herself at the hospital I could feel its point projecting through the thyroid cartilage under the tissues of the neck. I carefully cut down upon the point of the pin and seized it with a pair of forceps, introduced a tenotomy-knife along it to enlarge the wound in the cartilage so that its head could pass, and withdrew it without difficulty. The patient recovered without any untoward symptoms.[1]

The following case, which very recently came under my notice at the Children's Hospital, presents two conditions of interest, due to the presence of a foreign body in the trachea, which, in my experience, are very unusual.

Annie R., aged four years, was admitted to the Children's Hospital with marked dyspnœa, which was said to be due to the presence of a pin in her air-passages, which had been introduced some ten days previously. Prof. Ashhurst, under whose care the case was, opened the trachea below the isthmus of the thyroid gland, and as soon as the trachea was opened there occurred from it the most profuse hemorrhage, and with the blood there were expelled masses and shreds of fibrous material. The patient was in such a condition from the loss of blood that, as soon as the hemorrhage had ceased, it was deemed advisable to introduce a tracheotomy-tube, and, as she was breathing well, to postpone the attempt to remove the foreign body until she had reacted. Seven hours after the tracheotomy had been performed, she had another profuse hemorrhage, and in the absence of Prof. Ashhurst I was asked to see her. I found her breathing much obstructed, very little air passing through the tube. I removed the tracheotomy-tube, and with forceps extracted a large fibrinous cast (Fig. 14) from the trachea below the wound, after which her color improved and she breathed well through the wound. Upon carefully exploring the wound, I discovered the point of the pin lying close to the posterior wall of the trachea, and seized it with forceps and removed it. It proved to be a brass shawl-pin with a large head, more than two inches in length (Fig. 15). From its position I think the head was impacted in the right bronchus. The patient made a good recovery, and was discharged from the hospital two weeks after her admission, with the wound healed.

FIG. 14.



Fibrinous cast removed from trachea.

FIG. 15.

Brass shawl-pin (actual size).

The unusual conditions in this case were the profuse hemorrhage from the trachea, and the presence of large quantities of fibrous material closely resembling the membrane seen in cases of diphtheria. The source of the hemorrhage is in my mind a matter of uncertainty, but it may have arisen from the separation of the false membrane from the congested and inflamed mucous membrane of the trachea. The false membrane itself probably arose from the irritation set up by the presence of the foreign body in the trachea; but, in a number of cases in which the foreign body had remained in the trachea for some time, I have never seen a similar condition obtain, although the tracheal mucous membrane was much thickened by inflammatory swelling.

TRACHEOTOMY FOR PAPILLARY OR CYSTIC GROWTHS OF THE LARYNX OR TRACHEA.

Papillary or cystic growths of the trachea or larynx may occur during infancy or childhood, and may cause such urgent dyspnœa that tracheotomy may have to be performed to prevent death by suffocation, or it may be employed as a preliminary step in the treatment by intra-laryngeal methods or by thyrotomy. In children, according to Mackenzie,[1] the presence of these growths in the larynx is always attended with great danger to life, on account of the small size of the larynx: the tendency to spasm, catarrhal laryngitis, or laryngismus is very marked in this class of patients. When the growths cannot be removed by intra-laryngeal treatment and the dyspnœa is marked, no time should be lost in performing tracheotomy. The steps of the operation are those which have been detailed before, and need not be here repeated. A tracheotomy-tube should be introduced, and, after it has been worn a few weeks, attempts should be made to remove the growth by forceps introduced into the larynx through the mouth, or in some cases it can be operated upon from the tracheal wound. Occasionally a thyrotomy is required before the growth can be successfully removed, in which event the preliminary tracheotomy is of decided advantage.

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[1] Medical News, November 17, 1883, p. 541.

After the growths have been removed, and if there is no tendency to their recurrence, the tracheotomy-tube should be dispensed with as soon as possible. The tracheal wound may be allowed to heal, or the child may wear a shield with a nipple to keep the wound from healing for a time, so as to allow of the introduction of the tube if necessary.

If stenosis of the larynx or trachea from cicatricial contraction occurs after the removal of the growths, the use of a bougie or the introduction of an intubation-tube at intervals may be followed by good results.

TRACHEOTOMY FOR FRACTURES OR LACERATIONS OF THE LARYNX OR TRACHEA.

Fractures, lacerations, or contusions of the larynx and trachea are occasionally met with as the results of blows or falls upon the neck, and these injuries may be followed quickly by a dangerous form of dyspnœa from mechanical obstruction due to displacement of the fragments, the escape of blood into the air-passages, emphysema, and later from inflammatory swelling of the parts. Fractures of the larynx or trachea are not common in childhood. In Dr. Hunt's[2] collection of twenty-seven cases of this injury, only five occurred in children. Mr. Holmes[3] has recorded sixty-nine cases of fracture of the cartilages of the larynx and trachea, which gave fifty-three deaths and sixteen recoveries. Dr. Fussell[4] has reported a fatal case of fracture of the larynx and trachea in a boy, which was received while playing base-ball, the ball striking him upon the anterior surface of the neck.

If dyspnœa is observed, or if there is free bleeding, as evidenced by the expectoration, after such an injury, tracheotomy should be immediately performed, and a tracheotomy-tube should be introduced and worn until repair has taken place in the injured parts. If there is much emphysema, a tube longer than the ordinary one may be required. If there is free bleeding from the mucous membrane of the larynx, the trachea and larynx above the wound may be plugged with bichloride or iodoform gauze from the tracheotomy-wound: this will generally thoroughly control the hemorrhage, and will also keep the fragments in position. The mortality in this class of injuries is great, but it is decidedly less in cases in which tracheotomy has been performed.

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[1] Loc. cit., p. 314.

[2] Gross, System of Surgery, vol. ii. p. 317.

[3] Holmes, System of Surgery, vol. i. p. 749.

[4] Medical News, March 17, 1888.

TRACHEOTOMY FOR BURNS OR SCALDS OF THE LARYNX OR TRACHEA, OR FOR INJURIES TO THE LARYNX BY THE INTRODUCTION OF ACIDS OR OTHER CORROSIVE SUBSTANCES, OR IRRITATING GASES.

In children who have inhaled steam or hot air from a flame, a dangerous form of dyspnœa is rapidly developed, which is largely due to an acute œdema of the mucous membrane of the glottis, the larynx or trachea becoming secondarily involved. In such cases scarification of the parts may give relief; but, if it is not possible to do this, or if having done it the dyspnœa persists, tracheotomy should be promptly performed, and is the only means of saving or at least of prolonging life. Of fifty-one cases of tracheotomy reported by Durham[1] for this cause, thirty-five ended in death. The same indications exist for the operation to relieve the dyspnœa due to the introduction of irritating gases or acids or other corrosive substances into the larynx or trachea.

TRACHEOTOMY FOR GLOSSITIS OR MACROGLOSSIA, POST-PHARYNGEAL ABSCESS, OR TUMORS PRESSING UPON THE TRACHEA OR LARYNX, CAUSING DYSPNŒA.

Tracheotomy may be required in cases of glossitis or macroglossia, from the mechanical obstruction presented to the entrance of air into the larynx causing dyspnœa, or as a preliminary step in the operative treatment of these affections.

The operation may also be required for the same reasons in cases of post-pharyngeal abscess or tumors growing from the base of the skull or the anterior surface of the cervical vertebræ, projecting into the pharynx or œsophagus, and causing partial occlusion of, or pressure upon, either the larynx or the trachea, or from tumors having their origin in the neck and diminishing the calibre of either of these organs. In the latter class of cases the trachea may be much displaced, thus rendering the operation more difficult; and the surgeon should proceed with great caution, as the anatomical relations of the parts are often much disturbed.

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[1] Holmes's System of Surgery, vol. i. p. 754.

LARYNGOTOMY.

In the operation of laryngotomy the opening into the windpipe is made through the crico-thyroid membrane. It is a simple operation and one which is practically free from risk, and therefore can be performed much more rapidly and safely in an urgent case than tracheotomy. Although, from the ease of its performance, laryngotomy has had many advocates, yet it labors under some disadvantages as compared with tracheotomy, which have been pointed out by Mr. Marsh,[1]—namely, that the crico-thyroid space does not admit a sufficiently large tube, that the insertion of a tracheotomy-tube through the crico-thyroid membrane interferes with the integrity of the larynx and consequently the vocal apparatus may be damaged, and that serious inflammation or necrosis of the cartilages may result from the long retention of a tube in the crico-thyroid space. The first of these objections —that a sufficiently large tube cannot be introduced—is hardly to be accepted when we take into consideration the fact, which Mr. Holmes has pointed out, that the lumen of the cricoid cartilage is much greater than that of the glottis.

Laryngotomy is not applicable in young children, on account of the limited size of the crico-thyroid space in this class of patients. In cases of foreign body lodged in the larynx, it may be found a satisfactory operation; but in cases of diphtheritic croup or foreign body in the trachea or bronchus, in spite of the ease of its performance, I do not think it is as good an operation as tracheotomy.

In the operation of laryngotomy the same objection exists to the use of an anæsthetic as in that of tracheotomy, and therefore it had better be dispensed with. The patient being placed in the recumbent position, with the shoulders slightly elevated and the head thrown back to make the neck as prominent as possible, the surgeon feels for the prominence of the thyroid cartilage, and, steadying the larynx between the finger and thumb of the left hand, he makes an incision in the median line over the centre of the thyroid cartilage and extending downward for an inch or an inch and a half. The skin and superficial fascia being divided, the fascia between the sterno-hyoid muscles and the areolar tissue is exposed and divided, and the crico-thyroid membrane is exposed. The knife is then passed transversely through the membrane into the larynx, care being taken that both the membrane and the mucous membrane which covers its inner surface are divided at the same time, for Holmes refers to a case in which the tube was pushed down between this structure and the mucous membrane, the latter not having been perforated by the knife. As soon as the knife enters the cavity of the larynx, mucus and blood will be forcibly expelled. The wound should now be sufficiently enlarged and a tracheotomy-tube should be introduced and held in place by tapes secured around the neck. The tube used in cases of laryngotomy differs from the ordinary tracheal canula in that it is slightly flattened. The only troublesome bleeding which is likely to occur is from the crico-thyroid arteries or veins: if these cannot be avoided and are divided in the operation, they should be ligatured or temporarily secured by hæmostatic forceps, and, if the case is not extremely urgent, all bleeding should be arrested before the crico-thyroid membrane is incised.

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[1] St. Bartholomew's Hospital Reports, vol. iii. p. 338.

The after-treatment of cases of laryngotomy is similar to that employed in cases of tracheotomy: the same care is required in the attention to the tube and in the general management of the patient.

LARYNGO-TRACHEOTOMY.

The operation of laryngo-tracheotomy consists in dividing one or two of the upper rings of the trachea, the crico-tracheal membrane, the cricoid cartilage, and the crico-thyroid membrane. This operation may be employed in cases in which, from the age of the patient, the crico-thyroid space is too small to admit of a sufficient opening, or in those in which, for any reason, the surgeon does not deem it advisable to attempt to open the trachea lower down. The incision in the skin and superficial fascia of the neck is necessarily carried a little farther downward than in laryngotomy, and in dividing the trachea and cricoid cartilage the incision should be from below upward, to avoid wounding the isthmus of the thyroid gland, which may in some cases have to be displaced downward before the upper rings of the trachea are exposed. This operation is more often performed in the high operation of tracheotomy than is generally supposed, and it is open to the same objections which apply to laryngotomy. Holmes[1] mentions necrosis of the cartilages as liable to occur after this operation, and speaks of two fatal cases of this nature which have come under his observation.

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[1] Loc. cit., p. 773.

ATELECTASIS PULMONUM.

BY FRANCIS MINOT, M.D.

Synonymes.—Collapse of the lung; Fœtal condition of the lung, Apneumatosis.

Definition.—Atelectasis (imperfect dilatation) of the pulmonary air-vesicles is a condition in which the alveoli are empty and collapsed, their walls being in contact. It may be due to causes acting shortly before birth, or it may be acquired at any time afterwards, even in a portion of the lung which was previously expanded.[1] In cases in which no respiratory act has taken place, the whole of both lungs of course presents this condition. In the acquired form the extent of collapsed lung varies from a small area to one or more lobes.

History.—The more or less complete collapsed condition of the lungs sometimes found in new-born infants, which was formerly considered to be the result of pneumonia, was first shown by E. Jörg, in 1832, to be only a persistence of the normal fœtal condition, to which he applied the term atelectasis. Acquired atelectasis, that occurring after birth, was, however, still attributed to the effect of chronic pneumonia. In 1835 Rufz drew attention to an alteration of the pulmonary tissue (carnification) which he affirmed to be distinct from hepatization; but, as Willshire has pointed out,[2] the condition which we now call atelectasis was accurately described by Alderson in 1830, as found in the lungs in fatal cases of whooping-cough. Dr. Alderson[3] contrasted the appearances with those of hepatization; the individual lobules were more dense, of a dull red color, devoid of air, and sank in water, the tissue being dense and contracted, as if the air had been expelled and the sides of the air-cells agglutinated together. Moreover, there was no evidence of pleuritic inflammation. In 1844 Legendre and Bailly demonstrated that the atelectatic portions of the lung could be restored to the normal condition by insufflation, thus establishing the difference between atelectasis and the hepatization of pneumonia.

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[1] Acquired atelectasis is by no means confined to the period of childhood. It occurs in adults as a complication of typhoid fever and other debilitating diseases.

[2] Hughes Willshire, Historic Data on Infantile Pneumonia, in the Brit. and For. Med.-Chir. Review, Oct. 1853, p. 518.

[3] James Alderson, On the Pathology of Hooping-Cough, in Med.-Chir. Trans., vol. xvi. p. 78.

Etiology.—When from any cause the function of respiration fails to be established after birth, the lungs remain in a more or less complete fœtal condition; in other words, a state of pulmonary atelectasis exists, which may be the result of various causes, some of which are intrinsic, or pertaining to the child, while others are extrinsic and act upon it from without. Among the former are an imperfect fœtal development, as in premature delivery, or inherent feebleness due to ill health of the mother, or to disease inherited from her; from either of these causes the muscular power of the child is not equal to the effort of fully expanding the chest. There may also be imperfect development of the nervous respiratory centres of the fœtus, which do not then respond to the lack of oxygen resulting from the detachment of the placenta. In the latter case no effort at inspiration is made, and the child is still-born. The extrinsic causes include all those conditions which interfere with the supply of the maternal blood to the child, such as more or less complete detachment of the placenta, either before or during labor, as in placenta prævia, or frequent and violent uterine contractions in prolonged labor which arrest the circulation of the blood in the placenta. Breech-presentation often causes dangerous and fatal asphyxia to the child by compression of the umbilical cord or of the placenta between the fœtal head and the cervix uteri or the pelvic wall. Moreover, under these circumstances the sudden interruption to the supply of oxygen from the maternal blood excites violent inspiratory efforts on the part of the child, but in the absence of air he inhales whatever is presented to his mouth and nostrils, and hence liquor amnii, meconium, blood, mucus, or other foreign matters are liable to be drawn into the larynx, and may penetrate to the bronchia, or even to the air-cells.[1]

Acquired pulmonary atelectasis is most often observed in delicate or constitutionally feeble children, especially those who are rachitic, or whose vital condition has been lowered by insufficient or unsuitable nourishment, or by unhealthy sanitary surroundings, or who have been prostrated by exhausting disease, and are thus unable to resist the exciting cause of the affection, which in the majority of cases is catarrhal inflammation of the bronchial mucous membrane, especially capillary bronchitis, whooping-cough, diarrhœa, etc. The thick mucus accumulates in the smaller air-tubes, from which the enfeebled child is unable to expel it effectually by coughing. Access of the air to a larger or smaller number of lobules is thus prevented during inspiration, while any residuum of air remaining in the vesicles can still be driven out by expiration, or disappears by absorption, and the walls of the vesicles collapse. Gairdner, of Glasgow, first pointed out that the bronchial tube might be obstructed by a firm plug of mucus, which, acting like a ball valve, would allow the air to escape from the vesicle in expiration, but prevent its entrance during inspiration. But there are cases in which collapse of the lung occurs independently of any affection of the air-passages. West[1] mentions one of this kind in which the patient, a little girl five months old, died greatly exhausted from diarrhœa. There was extensive atelectasis of the right lung, but the bronchia were pale and contained no secretions.

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[1] Max Runge, Die Krankheiten der ersten Lebenstage, Stuttgart, 1885, p. 8.

Next to bronchitis, whooping-cough is perhaps the most efficient cause of pulmonary atelectasis in children, after which come measles, typhoid fever, and severe diarrhœa. A less frequent though not very rare cause of atelectasis in children is external compression of the lung from large pleuritic effusion, and sometimes from pericardial effusion. It may also arise from extreme curvature of the spinal column with diminution of the size of the thoracic cavity. The dorsal position of the patient not only favors the process of atelectasis, but is sometimes the actual cause of it in long-continued and prostrating diseases, such as typhoid fever, marasmus, etc.; and knowledge of this fact may be utilized in their treatment.

Pathology and Pathological Anatomy.—Literally speaking, pulmonary atelectasis is not a morbid condition: it is the normal state of the lung so long as the oxygenation of the blood is being carried on by means of the placenta. Hence no mention is made of it by some of the most eminent pathologists.[2] But in the process of evolution what was once normal may become afterwards pathological, and atelectasis after birth is as truly pathological as is patency of the foramen ovale. Without any inflammatory process, but simply through mechanical causes, the evolution of the lung is arrested, and it remains solid; or, when acquired after birth, it reverts to that condition after having previously become expanded.

The regions of the lung involved in atelectasis vary according to circumstances, but the morbid condition is usually found in the dependent and posterior portions, either as a single circumscribed area or involving a much greater extent of the organ, sometimes the whole of one lobe. Of course if the child have never breathed, both lungs are completely atelectatic. In slight cases only the edges of the lobes are collapsed. In atelectasis from bronchitis we often find the collapsed portions scattered over various parts of the lung in small areas, corresponding to one or more lobules which are implicated (the so-called lobular pneumonia). Under the same condition, as well as in cases due to extreme prostration, to marasmus, etc., the collapsed portion often occupies the postero-inferior margin of both lungs in the form of a broad streak parallel with the vertebral column, diminishing in breadth from below upward. In atelectasis from compression by pleuritic effusion the collapse involves the lower lobes of the lung in moderate cases; but in long-standing, abundant exudations the entire organ is usually implicated. As already stated, the situation of atelectasis in typhoid fever and other asthenic diseases is apt to be influenced by the position of the patient, corresponding to the side upon which he lies. The collapsed portions are chiefly confined to the surface of the lung, being seldom found in the interior except in cases of extensive invasion. The small areas of atelectasis resulting from local bronchial obstruction are wedge-shaped, the base corresponding to the surface of the lung.

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[1] Charles West, Lectures on the Diseases of Infancy and Childhood, 1874, p. 301.

[2] For example, Wagner, Handbuch der allgemeinen Pathologie, 1876.

Owing to their collapsed condition, the affected portions occupy less space than the normal lung-tissue: hence they are depressed below the level of the surface of the organ, and, when large areas of both lungs are involved, a greater extent of the heart's surface is thus exposed than normal. The collapsed areas, being composed of several lobules, are irregular in their outline. Their color is violet, reddish blue, or steel-blue, externally. On section the cut surface is dark red, and smooth, and a clear or sanguinolent fluid exudes from it, and from its resemblance to flesh it is said to be in a condition of carnification. It is firm to the touch, devoid of crepitation, dense, and not easily torn. It contains no air, and sinks in water. On inflation through the bronchus leading to the collapsed portion, the latter is at once restored to its normal appearance, unless the atelectatic condition has existed a long time, and chiefly in cases due to effusions of serum or pus in the pleura. When atelectasis occurs in connection with bronchitis, the mucous membrane of the air-tubes is softened, and red or pink in color, and the tube contains a thick muco-purulent mucus, which in the branches leading to the collapsed air-cells is sometimes inspissated, forming a complete plug.

The effect of extensive atelectasis on the organs of circulation sometimes shows itself in retarding or preventing the closure of the fœtal passages,—the foramen ovale, the ductus arteriosus, and the ductus venosus,—which in the normal condition is brought about by the establishment of the function of respiration. Incomplete expansion of the lungs has been considered a more or less important cause of delay in the obliteration of those passages, particularly of the foramen ovale and the ductus arteriosus, with the result of causing dilatation of the left auricle and thrombosis of the right ventricle, pulmonary artery, and cerebral sinuses.[1]

In proportion to the extent of the collapse of the pulmonary vesicles in acquired atelectasis we commonly find a compensatory dilatation of those which are still permeated by air, provided the child have sufficient strength in the inspiratory muscles. In feeble children emphysema rarely accompanies atelectasis.

In congenital atelectasis, especially after severe labors, and particularly after breech presentations, the right ventricle of the heart and the large vessels are distended with fluid blood. The sinuses of the dura mater and the vessels of the pia and those of the liver are also congested. Ecchymoses are observed on the serous membranes, and on the surfaces and even in the substance of the kidneys and other organs. Bloody serum is found in the pleural, pericardial, and peritoneal cavities. The larynx, trachea, and large bronchia may contain liquor amnii, meconium, or other foreign substances, when violent efforts at respiration have been made before birth. Some portions of the lung may occasionally be found distended by air which has penetrated into the uterus in consequence of the introduction of instruments or of the hand of the operator and has been inhaled by the child before delivery. Of course, if any respiratory efforts have been made after birth, the extent of dilated lung-vesicles would be correspondingly greater.

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[1] Gerhardt, Handbuch der Kinderkrankheiten, 3er Band, 2te Hälfte, p. 501.

Symptoms.—When from any of the before-mentioned causes the newborn child makes no efforts, or only feeble ones, at respiration, the limbs hang motionless, the eyes are closed, the skin is white, and the only sign of life is a feeble pulsation of the heart, perceptible to the finger. If a faint gasp be noticed at intervals, it is accompanied by a moist rattling sound. In favorable cases the effect of contact with the external air, together with the lack of oxygen due to the separation of the placenta from the uterine wall, aided perhaps by stimulating applications to the skin, excites a powerful inspiratory act, which is followed by a loud cry, and in a few minutes the function of respiration is established. The child opens its eyes and moves its limbs, and the integument assumes a bright rose-color.

In cases of more profound asphyxia the child is pale, the lips only having a bluish tint; the neck, the limbs, and the jaw are limp; the impulse of the heart is barely perceptible, and the respiratory efforts are slight or altogether absent. There is no reflex irritability, and the bodily temperature sinks. Unless restored by prompt and judicious treatment, the majority of children born in this condition quickly die, but in some cases life is prolonged for hours and even days.

Acquired atelectasis in children occurs most commonly in the early period of life, when diarrhœa, whooping-cough, bronchitis, etc., make their appearance. The symptoms relate chiefly to the respiration, and vary with the extent of lung-tissue involved. The rate of the respiration is increased, but the respiratory movements are shallow, the inspiration being slower and more difficult than the expiration. Should the area of pulmonary collapse be large, the remaining portion of lung is not capable of expanding to the extent of filling the vacant space created by the action of the inspiratory muscles, and the elastic chest-walls yield to the atmospheric pressure. This is shown by the sinking in of the intercostal spaces and supra-clavicular regions, and also by the retraction of the lower part of the sternum and lower ribs, giving rise to a deep furrow over the xiphoid cartilage and the sixth and seventh costal cartilages. The pulse is accelerated and its volume diminished in proportion to the amount of the consolidation. Cough is not caused by atelectasis, but the latter is very frequently the result of bronchial catarrh, which is accompanied by a persistent, moderate cough. The integument has a dusky hue, gradually deepening, in unfavorable cases, to lividity.

In well-marked cases the physical signs give evidence of more or less extensive solidification of the lungs, with catarrhal inflammation of the bronchial mucous membrane: hence the disease was for a long time confounded with pneumonia. Except, however, when a large portion of the lung-tissue is deprived of air, such as follows abundant pleuritic effusion, or pneumothorax, the dulness on percussion is often slight, and when the areas of solidification are limited in extent, and scattered, it may be absent. The dulness is often noticed in the lower and posterior regions of the chest, occupying a narrow margin near to and parallel with the spine, and extending upward. In the early stage of the affection, especially when complicating typhoid fever and other prostrating diseases, the situation and the intensity of the dulness may change according as the patient has varied his position, always seeking the lowest level.

On auscultation of atelectatic regions of limited extent, we notice only some diminution of the pulmonary vesicular murmur, with here and there a little moist crepitation. Where a larger area is involved, especially one surrounding a bronchial tube, bronchial respiration is heard, and sometimes a fine crepitant râle may announce an extension of the affection to neighboring air-vesicles. Under the same conditions bronchophony, varying in loudness according to the extent of lung-consolidation, is also manifest.

Atelectasis, not being an inflammatory condition, gives rise to no increase of bodily temperature, although it often complicates diseases which are accompanied with fever, such as broncho-pneumonia.

The general condition is that of prostration. In severe cases there are restlessness and sleeplessness. The child takes but little notice of anything, and makes but little complaint. There is no desire for food. The bowels are not especially disturbed, unless there be some intestinal disease. The duration of life under these circumstances is sometimes surprising; the child may linger for weeks and even months before it dies from exhaustion.

Diagnosis.—The atelectasis of new-born children due to deficient respiration may be recognized by the facts that the child is usually undersized, makes no voluntary movements of the limbs, and shows no other signs of life than an occasional faint, imperfect effort at inspiration, and a feeble pulsation of the heart. The labor may have been quite normal, if the symptoms are due to the inherent condition of the infant. Percussion of the chest reveals a more or less extensive consolidation of the lung, but the breathing is too feeble to afford trustworthy auscultatory signs. This condition is to be distinguished from asphyxiæ due to cerebral congestion or hemorrhage, or to imperfectly oxygenated blood, which are apt to occur in long and difficult labors, especially those involving compression of the umbilical cord or premature detachment of the placenta, or which are caused by the inhalation of foreign substances, as in breech-presentations, and which may be recognized by the progressive slowing of the fœtal heart, not only during an expulsive pain, when it is a normal phenomenon, but in the intervals between the pains, and also by the expulsion of meconium.[1]

Acquired atelectasis is likewise most common in feeble and poorly-nourished children, and is directly caused by any disease which interferes with the respiration or which favors pulmonary congestion. Hence it is liable to accompany capillary bronchitis, whooping-cough, measles, and severe and long-continued fevers, especially typhoid, which favor a dorsal decubitus. As it reveals itself by no striking symptom, it may be overlooked unless frequent examinations of the chest are made. The respirations are increased in rate and diminished in force, and there is progressive dyspnœa, with failure of strength. Pulmonary consolidations, if of any considerable extent, are recognized by dulness of the percussion-note, and frequently by bronchial respiration, but they are often of limited area, although they may be scattered over a large part of the lung, in which case the physical signs are inappreciable, as is usually the case in bronchial catarrh and in broncho-pneumonia. In atelectasis from dorsal decubitus they are very marked, and by an inexperienced observer might be mistaken for those of croupous pneumonia, but the characteristic temperature-curve of the latter disease is wanting.

If a large portion of the base of one lung were in a state of atelectasis, it might be mistaken for a pleuritic effusion; but in the latter condition bronchial respiration, bronchophony, and vocal fremitus would be absent, and the situation of the dulness might vary with the position of the patient. In a doubtful case puncture would settle the question.

Treatment.—In congenital atelectasis the most important element of the treatment consists in the employment of means calculated to arouse the dormant function of respiration, but in all cases the mouth and throat of the child should first be examined, to ascertain whether the air-passages be obstructed by foreign matters, which if found must be removed with the finger wrapped in a soft moist rag. In most cases in which there has been no especial complication or delay in the labor, simply blowing in the child's face, rubbing its chest and back with a towel, or slapping them with the corner of the towel wet with cold water, will be followed by a gasp and a cry, and the breathing is established. If these means fail, the Silvester method of resuscitation in drowning cases will often succeed. The child is laid upon its back, and both arms are slowly and simultaneously raised towards and alongside the head, and then replaced and pressed against the sides of the chest to expel the air from the lungs. This manœuvre is to be repeated gently from fifteen to twenty times a minute. It has been claimed that better results are obtained when the efforts at resuscitation are made in a cold room, and even when the child is laid naked on the cold floor, and Busey succeeded in restoring life under these conditions by the Silvester method in two apparently hopeless cases.[2]

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[1] Runge, loc. cit., p. 7.

[2] A System of Obstetrics by American Authors, 1888, vol. i. p. 519.

The method suggested by Schultze has in the experience of the writer been very efficient. The child, being laid on its back with its head towards the operator, is grasped by the hands applied to its chest and shoulders in such a way that the head falls backward, the face towards the knees of the operator, while the belly and legs hang down in front. The weight of the head in one direction and of the rest of the body in the other causes an enlargement of the chest by traction, with depression of the diaphragm, and promotes inspiration. The operator then swings the child quickly upward, reversing its position, so that the head is flexed upon the chest, while the trunk and legs fall downward and towards the face, thus compressing the chest and expelling the air.

Faradization may also be tried with caution. A large, well-moistened sponge electrode should be applied to the nape of the neck, or other part of the child's body, the other pole being brought in contact for a moment only with the skin behind the lower part of the sterno-cleido muscle, so as to stimulate the phrenic nerve. A deep inspiration will take place, and the lungs will expand, after which the lower part of the chest must be carefully compressed to empty the lungs again.[1] As in all other methods, the proceeding must be repeated from twelve to twenty times a minute, until independent respiration is established. Care should be taken that the current be only strong enough to cause a sufficient inspiratory effort. In all cases the infant should be watched for some time afterwards, since there is danger of relapse in feeble children.

The treatment of acquired atelectasis is closely connected with that of the diseases of which it is the result, especially bronchitis, whooping-cough, broncho-pneumonia, and typhoid fever. It will therefore here be considered only from a general point of view, the reader being referred to the articles on those subjects for more precise information. The underlying cause of atelectasis is debility, and the indications for a supporting and stimulating treatment are evident. Pure air and free ventilation, good sanitary surroundings, and proper nourishment are of the first importance. The removal of the patient from the city to the country, especially to an elevated region, or to the sea-shore, is often of great benefit. A prophylactic treatment can sometimes be instituted with advantage, in diseases which are accompanied by much prostration and in which the patient is liable to remain for a long time on his back, by changing the child's position alternately from one side to the other, supporting it with cushions or pillows. The condition of the lungs should be ascertained by auscultation and percussion as thoroughly as the patient's strength will permit, at least daily, in order to meet the earliest indications. The diet should be nutritious, and as acceptable as possible, and food should be given at frequent intervals, care being taken not to overload the stomach. Milk, gruel of barley and other farinaceous substances, the various malted foods, chicken and veal broths, plain ice-cream, cooked fruit, such as roasted apples, and a liberal supply of water, with occasional stimulants, especially good brandy, are the chief articles of diet needed. The external application of cold water by means of sponging, and even the cold bath, judiciously employed, which is praised by Gerhardt,[1] may often be used with advantage. If the breathing becomes faint from exhaustion, the child should be enjoined from time to time to draw a full breath, in order to expand the lungs, and, if he fail to respond to the appeal, an external stimulant, such as cold water or electricity, may be tried.

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[1] A. Jacobi, Therapeutics of Infancy and Childhood, Archives of Pediatrics, March, 1888, p. 133.

The internal treatment consists mainly of stimulants, of which brandy is the most efficient. It is surprising how much of it may be given not only without injury but with positive benefit, under these circumstances, even to the youngest children. Jacobi[2] recommends from one to four drachms of brandy daily, with camphor-water, to young infants, and in desperate cases a five-hundredth of a grain of nitro-glycerin, to be repeated, if necessary, after fifteen or thirty minutes. The doses of these remedies may be increased in proportion to the age of the child. Carbonate of ammonium, in doses of from three to six grains, in sweetened water, every four hours, will also be found useful.

The general treatment of the patient is important. He should be disturbed as little as possible in the necessary arrangements for his care. Even the bath should be employed with caution if it be followed by fatigue. The room should be kept cool, and all persons should be excluded from it whose presence is not necessary. A good nurse, preferably a trained one, would of course add much to his comfort as well as to his chances of recovery.

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[1] Loc. cit., p. 511.

[2] Loc. cit., p. 135.

CROUPOUS PNEUMONIA.

BY FRANCIS MINOT, M.D.

Definition.—A specific, infectious, self-limited disease, giving rise to definite temporary pulmonary lesions. It is non-contagious, and its duration is from five to ten days.

Synonymes.—Pneumonia, Pneumonitis, Lung fever, Lobar pneumonia.

History.—It was not until the end of the first quarter of the present century, in 1823, that the distinction between bronchitis and pneumonia in children was first pointed out by Léger, and the latter disease was for many years afterwards confounded with pulmonary collapse, a condition described by Jörg in 1832 and 1835 and by him called atelectasis. About the same time Rufz affirmed atelectasis to be distinct from hepatization, suggesting that it might be simply the result of compression of the pulmonary tissue; while Rilliet and Barthez in 1838 and A. Reese in 1839 were inclined to consider it as the result of chronic pneumonia. But the interesting discovery made in 1844 by Legendre and Bailly, that the collapsed lung could be restored to the normal state by inflation, proved conclusively that atelectasis and hepatization were quite different pathological conditions, the latter only being the result of an inflammatory process, and called pneumonia. The distinction between lobar and lobular pneumonia (broncho-pneumonia) was first definitely established by Rilliet and Barthez in 1851. The gradual advance of medical science, and especially the accumulation of accurate observations on the course, range of temperature, etiology, and pathology of pneumonia, substantially led the way to a general belief that the disease was not of an inflammatory but of an infectious character, and this opinion, first formulated in 1872 by Juergensen, is now accepted by the most eminent clinical observers. Still later, certain micro-organisms capable of being cultivated, and, it is stated, of communicating the disease to animals by inoculation, were discovered by Klebs, Eberth, Koch, Friedländer, Fränkel, and others in the lungs of patients who had died of pneumonia. Whether the power of conveying the disease is confined to a single specific organism or is shared by more than one, is not yet determined.

Etiology.—So long as pneumonia was looked upon as a purely inflammatory disease its principal cause was naturally believed to be exposure to cold, and this belief was confirmed by the fact that it prevails most extensively during the winter and spring months, about two-thirds of all the cases occurring in this portion of the year, while only about one-third are observed in summer and autumn. The cough which accompanies pneumonia also readily suggests exposure, and the fact that the patient had "caught cold" was usually considered sufficient to account for his sickness. The disease, however, is not more common in cold climates than in temperate regions, and it not unfrequently occurs without any exposure, which shows that cold must be considered as a predisposing or exciting rather than an essential cause.

It was formerly believed that lobar pneumonia was rarely met with among young children; but more extended observation has shown that it is, on the contrary, one of the most common of the severe diseases of childhood. It is occasionally met with in infants at the breast, and its frequency increases from the end of the first year, the maximum occurring between the ages of four and seven. So far as statistics show, there is a slight preponderance in the number of male children who are attacked. Children as well as adults are somewhat more liable to a recurrence of the disease after having had a previous attack. Baginsky[1] mentions the case of a child six months old, in which measles followed pneumonia and was succeeded in its turn by a second and fatal attack of pneumonia. In cases reported by him, recurrence happened after eleven days, after three months, after a year, and after three years. In the case of a boy five years old under my care, the disease recurred three months after the first attack. The house in which he was living was not in good sanitary condition, and several other children of the same family had pneumonia at various times for two or three years, until the drainage was repaired.

Healthy children are quite as liable to be attacked by pneumonia as those who are delicate or cachectic, if not even more so.

The infectious nature of croupous pneumonia, now so generally admitted, throws much light upon the etiology of the disease, and explains its not unfrequent occurrence in dwellings whose sanitary condition is bad, especially where owing to defective plumbing there is a direct communication with a common sewer, privy-vault, or cess-pool; also in filthy and ill-ventilated tenement-houses, crowded jails and workhouses, etc. The following example of local endemic pneumonia, which came under my observation, is an illustration of this. A healthy male child, twenty-one months old, was attacked with croupous pneumonia, January 5, 1885 (see Chart I.). The house in which he was living was a new one, presumably in perfect sanitary condition, in a healthy part of Boston. In a closet opening into the child's nursery was a "set basin," communicating with the soil-pipe. The case was a mild one, and the boy was convalescent in five days. Meanwhile his nurse was suddenly taken with the same disease, and died in a few days. As she was too ill to be removed from the house, she was taken care of by her mother, an elderly woman, who soon showed symptoms of pneumonia, returned to her own home, and also died shortly afterwards. Lastly, a female infant, four months old, sister of the boy, in perfect health, and nourished at her mother's breast, sickened, January 9, with pneumonia of the apex of the left lung, and died on the 16th. (See Chart II.) An examination of the premises showed that owing to a defect in the soil-pipe there was an accumulation of filth upon the cellar floor, and also a free escape of sewer-gas into the house. In a prison in Amberg, in Bavaria, an epidemic of pneumonia occurred in 1880 in which one hundred and sixty-one persons were attacked. Pneumonic cocci were found in the stuffing of the mattresses, were cultivated, and successfully inoculated into animals.[1] Such facts show that the germs of pneumonia may enter our dwellings, and under unfavorable sanitary conditions may multiply there and communicate the disease to the occupants. In what way the micro-organisms gain access to the lungs is not certainly known, but probably it is by inhalation.

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[1] Dr. Adolf Baginsky, Practische Beiträge zur Kinderheilkunde, 1880, Heft i. p. 8.

When several cases of pneumonia occur in the same house, the idea of contagion naturally suggests itself; but there are no grounds for the belief in its direct transmission from one individual to another, and most if not all authorities agree that it is not contagious in the ordinary sense of the word.

Pathology and Pathological Anatomy.—Croupous or lobar pneumonia, which was for a long time looked upon as a purely local inflammatory affection of the lungs, is now generally regarded as a specific infectious disease, having a local pulmonary manifestation, caused by the invasion of the system by one or more micro-organisms of different forms, to say the least, which multiply after the manner of a ferment. In other words, pneumonia is a zymotic disease in the sense in which small-pox, typhus and typhoid fevers, cholera, etc., are zymotic. Whether the germ be unique, or whether the disease can be excited by more than one micro-organism, or, lastly, whether certain organisms differing in appearance but all effective may not be identical, though in different stages of development, is not determined; but it appears to be certain that in the great majority of cases the lancet-shaped encapsulated coccus of Fränkel is found in the tissues of the pneumonic lung (in seventy-eight out of eighty-three observations, or more than ninety-three per cent., according to Weichselbaum), and when inoculated into rabbits it conveys to them the disease. What is extraordinary is that, according to Fränkel, the same coccus is always present in the saliva of healthy human individuals, and that rabbits inoculated with human saliva die in the course of one to two days, the cocci being found in their blood.[2] Another remarkable fact is that the same micrococcus which is supposed to develop pneumonia may also be associated with cerebro-spinal meningitis. The two diseases are, in fact, sometimes clinically related, one graduating into the other.[1]

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[1] Ueber die Actiologie der fibrinosen Pneumonie, von Dr. Wilhelm Wolf, Wiener Med. Presse, December 25, 1887.

[2] Wolf, loc. cit.

The pathological anatomy of pneumonia in children does not differ from that in the adult. It embraces the stages of hyperæmia or congestion, of solidification or hepatization, and of softening or liquefaction. To these must be added suppuration and gangrene, which are rare in children, only occurring in the severest and usually fatal cases. The three principal stages are often found existing at the same time in the affected organ, showing that the disease was extending at the time of death, and this enables us to trace the different phases of the morbid process. The situation of the disease in children varies in the following order of frequency: 1st, the right apex; 2d, the left apex; 3d, the left base; 4th, the right base. Sometimes, of course, more than one region is occupied by the disease at the same time. The central portion of a lobe only may be involved, especially in pneumonia of the apex.

In the first stage, that of engorgement, the affected part is more voluminous than in the normal state, is of a dark-red color, and of a doughy consistence. It contains a diminished amount of air, sometimes none at all, and there is little or no crepitation on handling. A turbid, bloody serum flows from the cut surface. This condition approaches gradually the stage of hepatization, the alveoli becoming filled with an exudation containing an abundance of cells and an increasing amount of coagulated fibrin. The hepatized tissue is of a brick-red color. It is considerably swollen, and often bears the impression of the ribs on its surface. The cut or torn surface shows multitudes of little elevations projecting from it, consisting of the alveoli distended with the viscid exudation, and from this appearance, resembling that of a section of liver, its name is derived. The transition into the third stage is characterized by an induration marbled with various tints of yellow and gray, from a section of which flows a reddish-gray or milky exudation, due to a diminution of the capillary hyperæmia and an increase of the fibrin, together with fibrinous casts of the alveoli. The casts, together with the cells contained in the exudation and the alveolar epithelium, rapidly undergo a fatty degeneration, which during life favors their removal by absorption or by expectoration, after which the alveoli, becoming again permeable to the air, are gradually restored to their normal condition. Under unfavorable conditions hepatization may undergo a change into purulent infiltration, the granulations disappear from the cut surface, the lung-tissue becomes baggy, and is easily torn. Restoration, however, is possible in this condition, though often long delayed. Gangrene is a still rarer termination of the pneumonic process, resulting chiefly from thrombosis of the nutrient vessels. When limited in extent it may become surrounded by a wall of connective tissue, and gradually be eliminated by abscess-formation.[1] The lining membrane of the bronchia communicating with the hepatized region is softened and red, and the air-tubes contain more or less of the inflammatory exudation.

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[1] See "A Case illustrating the Relationship between Cerebro-Spinal Meningitis and Pneumonia," by Henry Hun, Albany Med. Annals, Aug. 1888; Townsend, Endemic Cerebro-Spinal Meningitis, Bost. Med. and Surg. Journal, July 19, 1888.

The pleura corresponding to the pulmonary lesion is usually more or less involved. In cases of only moderate severity there is ecchymosis or injection of the membrane; in severe ones, exudation of plastic lymph or of serum. The costal and pulmonary surfaces may become adherent at the spot. Sometimes the situation of the pleural inflammation does not correspond with that of the pneumonia.

Symptomatology.—Croupous pneumonia in children is an acute disease of brief duration, seldom lasting longer than ten days, and often completed in five or six. In many cases it is a primary affection, occurring suddenly while the patient is in good health. Its course consists of three periods, like that of most zymotic diseases,—the effervescence, occupying one or two days; the fastigium, embracing rarely more than three days; and the defervescence or crisis, often accomplished in one day, sometimes in two, but occasionally taking a more deliberate descent, or lysis, as in the case of Arthur D. (Chart IV.) There are premonitory symptoms in a considerable number of cases, such as cough, pain in the side, drowsiness, loss of appetite, or chilliness, which may be noticed for a day or two before the attack, but, on the whole, the absence of well-marked prodromal symptoms is as noteworthy a feature of the disease in children as in adults. The rigor which marks the beginning of the attack in adults is rarely seen,[2] but in place of it vomiting is usually observed, or, in the case of very young children, convulsions. Fever, with hot skin, restlessness, rapid pulse and breathing, and elevation of temperature, quickly follows. During the short period of effervescence the temperature frequently attains the highest point observed throughout the course of the disease, usually between 103° and 105° F. (Charts II., VI., VII.) So rapid is this increase of temperature that it may have reached its acme when the physician is first called to see the case (Charts I., VI., VII., VIII., IX.), and even when no prodromic symptom had been noticed.

The duration of the second stage, or fastigium, varies between two and five days, during which the condition of the patient undergoes but little change. The morning temperature in the axilla is between 102° and 104° F., the evening temperature between 104° and 106° F. The cough continues as before: it is suppressed as far as possible, but is rarely urgent. A deep flush is noticed on one or both cheeks, and an herpetic eruption is often seen on the lips. There is no appetite, and the child refuses everything but water and sometimes a little milk. The breathing is hurried, and the nostrils dilate with each inspiration.

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[1] L. Thomas, Croupöse Pneumonie, in Gerhardt's Handbuch der Kinderkrankheiten, 3er Band, 2te Hälfte, p. 602.

[2] Henoch has occasionally observed a rigor in the beginning of croupous pneumonia in children over five years old. Vorlesungen über Kinderkrankheiten, 1881, p. 330.

CHART I.

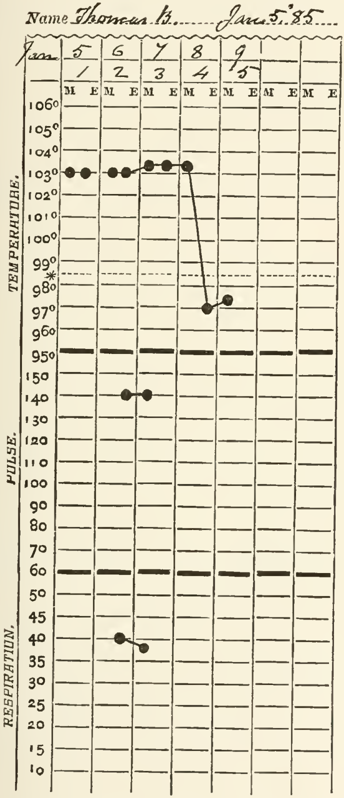

CHART II.

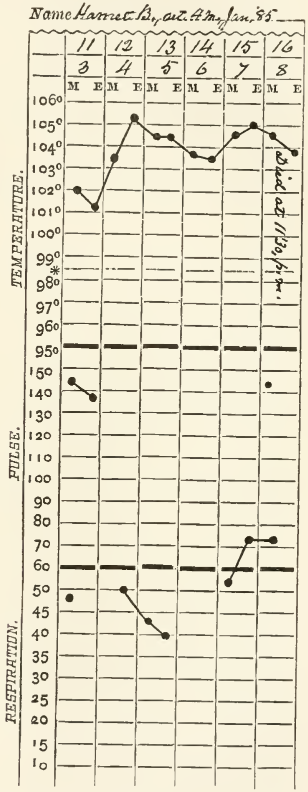

Chart I.—T. B. (boy), aged one year and nine months, January 5, 1885. The notes of this case were mislaid, and cannot be found. The chart shows that it was one of moderate severity.

Chart II.—H. B. (girl), aged four months and thirteen days. Previously healthy. January 8 and 9, 1885, feverish, crying frequently, nurses well. January 11, bronchial respiration in left apex; no severe symptoms till the 15th, then very restless, with frequent vomiting; 16th, universal jaundice, no urine for twelve hours, died at 11.30 P.M.

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The third stage, that of defervescence, is apt to begin suddenly, usually during the night, but sometimes it occurs during the day, as in the case of Arthur D. (Chart IV.) The fall often takes place within twenty-four hours, from the highest point to the normal, or more frequently to half a degree, a degree, or a degree and a half below it, where it may remain with slight fluctuations for a day or two after convalescence has begun (Chart IV.). But, although the defervescence is usually by crisis, in a few cases it is gradual, as in that of Joseph C. (Chart VI.), and occasionally it is irregular (case of George D., Chart IX.). With the fall of the temperature the skin becomes moist, and the pulse and the respiration show a corresponding improvement, which is also seen in the general appearance of the patient. His apathetic state is changed for one of cheerfulness, and he demands food for the first time.

Physical Signs.—The physical signs of pneumonia are the same in children as in the adult, but there is often some difference in their situation, corresponding to that of the seat of the lesions, which in children are much oftener found at the summit of the lung. In not a few cases, moreover, no satisfactory results are obtained by auscultation and percussion until a comparatively late period, owing to the limited extent of the affected region, and its position in the centre of a lobe, surrounded by healthy lung-tissue. The general symptoms, and especially the temperature-curve, will make the diagnosis of pneumonia almost certain in cases in which its locality may be ascertained only with considerable difficulty. This is shown in the case of Arthur D. (Chart IV.), a little boy three years old, who became drowsy and stupid, with complete loss of appetite, on the afternoon of March 25, 1887. The temperature-curve, as shown by the chart, pointed unmistakably to pneumonia, but it was not until the sixth day, when the crisis was almost completed, that dulness and bronchial respiration, though repeatedly sought for, were found in the right upper back. Fine crepitant râle is less often heard in the pneumonia of children during the early stage than in adults, though a moist râle is frequently present; but while the area of the disease is spreading we may hear it on the confines of the solidified region, though even there it is often mingled with the subcrepitant and also with pleuritic friction-sounds. True bronchial respiration is very clearly heard during the stage of hepatization, after which it is replaced by moist crepitation. I can confirm the observation of Emmett Holt,[1] that "the frequency with which the apex is involved should be borne in mind, and the region high in the axilla carefully examined. It is not infrequently the first, and may be the only, place in which bronchial breathing is heard."

In the early stage of the disease the indications furnished by percussion are sometimes more satisfactory than those by auscultation, though when a layer of elastic lung-tissue lies between the seat of the disease and the surface a somewhat forcible blow may be necessary to bring them out. A light percussion-hammer tipped with rubber answers well for this purpose. Of course the finger of the observer should be laid over the part, to receive the blow. Dulness will often be found to precede the bronchial respiration by several days, and it can be detected for a considerable time after the other signs of solidification have disappeared.

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[1] New York Medical Record, February 14, 1885.

Vocal resonance, especially that of the cry, is usually well marked during the stage of hepatization, and even before it. Vocal fremitus may also be perceptible to the hand laid over the seat of the disease, but its presence is very uncertain. In some cases in which auscultation and percussion are difficult or impossible on account of the condition of the child, the fremitus, if felt on only one side, may be of value in diagnosis.

A few of the most important symptoms demand especial notice. The general condition of the patient is that of apathy from exhaustion. He seems to have no breath to spare for complaint, and hence submits to physical examination with much less opposition than is often displayed by sick children. He will lie for hours making very little complaint, often grasping some toy, which he holds day and night. The complete absence of appetite in children with pneumonia, although very striking, seems to have attracted but little attention; at least but few writers allude to it.[1] During the period between the attack and the fall of temperature the child not only does not ask for food, but he refuses to take it so far as lies in his power, often going four, five, or six days without nourishment of any kind, but drinking water from time to time. The digestion is arrested, and after the stomach is emptied by vomiting nature tries to keep it so by taking away the appetite.

The pain in the side is rarely urgent, and may be wholly absent. It is excited by drawing a long breath and by the cough, which on this account is suppressed as much as possible. The pain does not always coincide with the seat of the pulmonary lesion, and is occasionally referred to the epigastrium. Its cause is supposed to be a concomitant pleuritic inflammation, which, however, must generally be very limited in extent, since the pain almost always disappears before the temperature falls to the normal. The explanation would not apply to such a case as that of George W. (Chart V.), who complained of pain in the right side, while the signs of consolidation were found only in the apex of the left lung; nor to those in which the pain is referred to the abdomen, or to the legs, as in that of George D. (Chart IX.) The attitude of the patient varies with the situation of the disease. He prefers to lie on the same side as the affected lung, which is more immovable in that position, so as to breathe more freely with the other. But he often lies on the back, with the hands sometimes raised to the head. The deep flush on one or both cheeks is rarely absent, and hence is of value as a diagnostic sign, but when on one side only it does not necessarily correspond with that of the affected lung.

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[1] Among them, Thomas, loc. cit., p. 651.

CHART III.

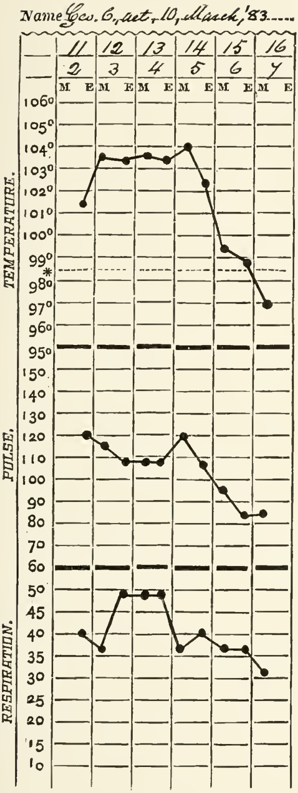

CHART IV.

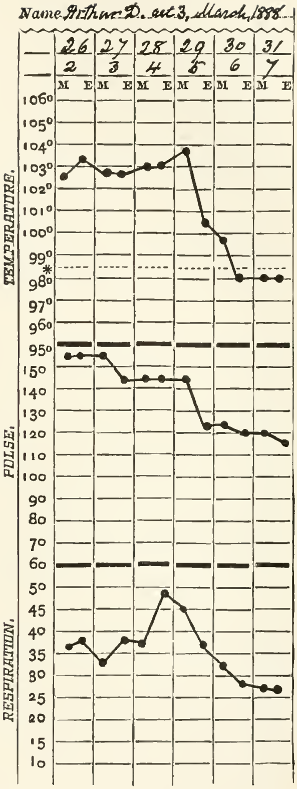

Chart III.—George C., aged ten years. A "cold" for a few days, but was out on March 9, 1883; 10th, vomiting and increase of cough, went to bed at noon; 12th, pain in left side, dulness and fine crepitant râle between angle of left scapula and spinal column; 13th, bronchial respiration in same place, moist râle below it; 14th, same signs extending upward, lower left back free; 15th, no bronchial respiration nor râle heard, tough reddish expectoration; 17th, convalescent. He took scarcely any nourishment till the 16th. (Solution of acetate of ammonium; Dover's powder.)

Chart IV.—Arthur D., aged three years. Previously well. In the afternoon of March 25, 1888, became drowsy and stupid and refused food; some cough, no signs found in chest; remained in the same state, taking no food, till the 29th, when he took an ounce of orange-juice, and continued the same, night and morning; on the 30th, dulness and bronchial respiration were found for the first time in the right upper back; 31st, bronchial respiration disappearing, and replaced by moist râle; April 1, convalescent.

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Expectoration is hardly ever seen in young children, who instinctively swallow the sputa. In the case of George C., ten years old (Chart III.), it was noted that there was a "scanty, tough, reddish expectoration."

The pulse during the fastigium is very rapid, being seldom less than one hundred and twenty, and often one hundred and forty, one hundred and fifty, and one hundred and sixty, in the minute. The rate of the breathing is also increased, and out of proportion to that of the pulse, so that, instead of the normal ratio of one respiratory act to about four and one-half cardiac pulsations, it is not unusual to observe one respiration to two and eight-tenths pulsations, to two and six-tenths, to two and three-tenths, etc. This change of ratio is not pathognomonic of pneumonia; it may occur whenever there is fever with rapid diminution of the respiratory surface, as in large pleuritic effusions; but in pneumonia it is of special value in diagnosis, because it begins before the other signs appear and lasts till after they have ceased.

Nervous symptoms are not rare in children with pneumonia. They are more common among younger subjects, but are by no means confined to them. Occasionally they are so predominant as to give rise to the belief that the disease is complicated with meningitis, the so-called cerebral pneumonia. It is also alleged by many authors that these symptoms are especially apt to be associated with disease of the apex of the lungs; but this is denied by Eustace Smith[1] and by Emmett Holt.[2] They are most marked at the height of the disease, and vary from mild delirium to actual mania, so that the patient can with difficulty be kept in bed. Violent symptoms, however, are not common. Persistent drowsiness or semi-stupor is not unfrequent.

In cases which terminate favorably the cessation of the severe symptoms is almost always rapid. The sudden fall of the temperature to the normal point—generally, indeed, to one or one and a half degrees below it—which occurs between the fifth and seventh day (cases of Thomas B., Chart I.; George W., Chart V.; Henry D., Chart VII.), and is accompanied by a corresponding amelioration of the general condition, is a striking characteristic of croupous pneumonia. The delirium and restlessness are followed by tranquil sleep, the pulse and respiration approach the normal rate, the pain in the side ceases, the skin is bedewed with perspiration, and the child for the first time asks for food. In exceptional cases the recovery is more gradual, as in that of George D., who had two attacks during the same season (Charts VIII. and IX.), the first of which was of the usual brief duration. In the second attack, three months later, a slight extension of the infiltration probably took place on the third day, the whole duration being eight days. The situation of the disease in both attacks was in the left lower lobe, behind.

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[1] Clinical Studies of Diseases in Children, 1887, p. 56.

[2] Cerebral Symptoms in the Pneumonia of Children, N. Y. Med. Rec., April 7, 1888.

Secondary croupous pneumonia may complicate various diseases, especially pulmonary tuberculosis, bronchitis, whooping-cough, measles, and typhoid fever. To the symptoms of the original affection are added those of the invading disease, of which the most conspicuous are rapid breathing and pulse, high temperature, and prostration. The physical signs are usually those of consolidation, followed by purulent infiltration, the earlier stage of hyperæmia being unnoticed on account of its short duration. The course of the disease thus complicated is usually rapid in children, as well as in adults, and the majority of the cases are fatal. Pneumonia itself may likewise be complicated with an intercurrent disease which adds greatly to its danger, such as bronchitis, measles, diphtheria, diarrhœa, whooping-cough, or a large pleuritic effusion. It is easy to understand that while a considerable part or even the whole of one lung may be deprived of its function by pneumonia without causing serious embarrassment to the respiration, provided the other lung be intact, the invasion of bronchitis in the latter would seriously imperil life.

Diagnosis.—The diagnosis of croupous pneumonia in children is not difficult in most cases. The important points to bear in mind are the acute nature of the disease and its sudden onset, the characteristic temperature-curve, the hacking cough, the fremitus of the cry, the increased rate of the respiration compared with that of the pulse, the dilatation of the nostrils during inspiration, and the physical signs furnished by auscultation and percussion.

The general condition of the child is strongly suggestive of the disease. From a state of health he quickly becomes apathetic, somnolent, stupid, and often delirious. Having no breath to spare for struggling, he submits to the physical examination by the physician with a docility which he might not exhibit in health. "In any case where an infant screams loudly during an examination of the chest," says Eustace Smith, "the probabilities are strong against the lungs being seriously diseased." In some cases we are obliged to depend upon the temperature-curve and the rational symptoms before satisfactory evidence of lung-consolidation is afforded by the presence of bronchial respiration, which sign may be delayed, owing to the central situation of the hepatized tract, until the beginning of the crisis (Chart V.), or even till the temperature has fallen to the normal point (Chart IV.). The general symptoms, however, are so characteristic that there is usually but little doubt. But in most cases on examination of the chest there will be found dulness on percussion in a limited district, often in one apex, and generally behind, together with crepitant râle, which soon gives place to bronchial respiration.

In the diagnosis of croupous pneumonia the presence of certain other diseases must be excluded, especially catarrhal or broncho-pneumonia, acute gastro-intestinal catarrh, and acute meningitis. Broncho-pneumonia may easily be confounded with true pneumonia by one who is ignorant of the distinctive symptoms of the two diseases, but an error of diagnosis could

CHART V.

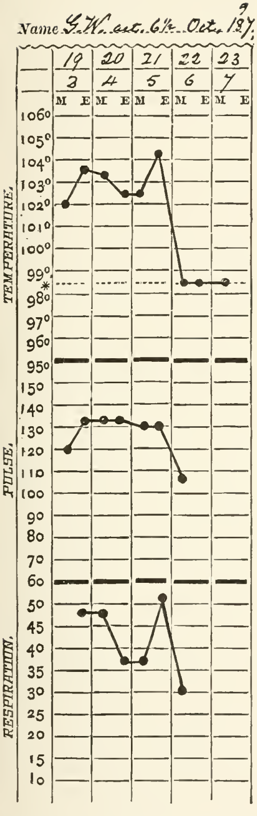

CHART VI.

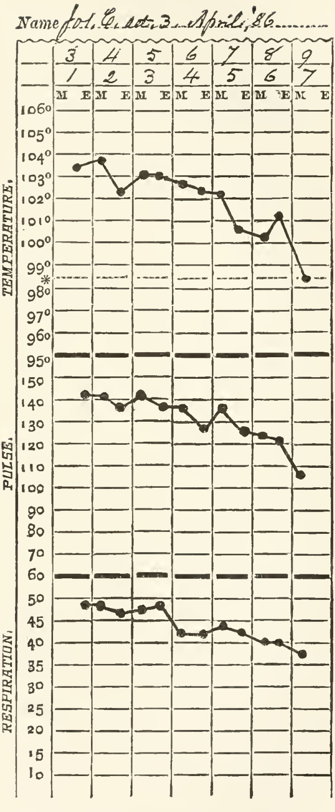

CHART V.—George W., aged six years. Cough for two days; October 17, 1879, vomited in the night; 18th, pain in *right* side; 19th, languid, hot skin, no appetite; no signs found in chest until the 21st, then bronchial respiration above spine of *left* scapula; 22d, he was free from symptoms.

CHART VI.—Joseph C., aged three years. Began to cough April 2, 1886; on the 3d, fever, hot skin, etc., prostration, lower posterior region of left lung affected; he took no food till the 10th. (Brandy and carbonate of ammonium; Dover's powder.)

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hardly be made by a competent observer. Croupous pneumonia is almost always primary, attacks healthy children, and has a brief duration and a definite range of temperature. The disease is usually limited to a single lobe, at least in the beginning of its course. Broncho-pneumonia is secondary to bronchitis, measles, whooping-cough, and other debilitating affections, is indefinite in its course and duration, involves both lungs, and has no characteristic temperature-curve. Râles in croupous pneumonia belong to the early stage, disappear after consolidation, and reappear when resolution begins. In broncho-pneumonia consolidation occurs in diffused limited areas, comes later, or may not come at all. "If the pneumonia is primary, and at the apex only, it can be pronounced lobar without hesitation."[1]

The vomiting which often marks the onset of pneumonia in children is sometimes urgent, and if accompanied by diarrhœa may resemble an attack of acute gastro-intestinal disease. In a doubtful case the temperature and the state of the lungs should be carefully watched.

Croupous pneumonia sometimes begins with active nervous symptoms, which, indeed, are occasionally prominent throughout its course, and, if the pulmonary symptoms are not obvious, the case may be mistaken for one of acute meningitis. But acute idiopathic meningitis is one of the rarest of diseases. In children it is usually secondary to disease of the middle ear or of the mastoid cells, and if careful examination excludes the existence of such disease the probability of cerebral complication is slight. Moreover, severe pain in the head is a prominent symptom of meningitis, and is rarely absent. Tubercular meningitis would be excluded by the absence of the prodromal period of irritability of temper or mental depression, of headache, of constipation, and especially of slow and irregular pulse. Careful attention to the temperature and the physical signs will determine the presence or absence of pneumonia, and in the former case the cerebral phenomena must be considered as symptomatic of that disease.

Prognosis.—Primary croupous pneumonia in healthy children is not only rarely fatal, but does not tend to leave behind it any permanent damage to the lung. The popular opinion that it is one of the most dangerous diseases of childhood is due, no doubt, to its being often confounded with broncho-pneumonia, a much more serious malady. In judging of the prognosis it is important to take into account the general condition of the child, his previous health, his surroundings, and any other diseases of which the pneumonia may be a complication or the sequel. The hygienic conditions under which it is so apt to make its appearance have hardly received the attention which their importance warrants. It is surprising how little they are alluded to in connection with etiology and prognosis and even treatment by writers on children's diseases. The possibility of removing children who are or who may become sick with pneumonia from bad sanitary conditions to better quarters is an element in the prognosis which should never be overlooked. Baginsky[1] states that out of sixty children with pneumonia, nearly one-half of whom were under two years of age, there were four fatal cases. Excluding nine which he was unable to follow out, there remained fifty-one, with four deaths, but of these four only one appeared to have been in good health before the attack. It was formerly supposed that the disease was much more fatal in young children than in older ones, but the contrary experience of Baginsky in this regard has been corroborated by numerous observations. It has also until lately been an accepted belief that pneumonia of the upper lobes was more apt to be accompanied by cerebral, and consequently dangerous, symptoms than pneumonia of the lower lobes. My own experience coincides with that of Eustace Smith and Emmett Holt, which I have already quoted, that there is really no difference in this respect. The cases of Arthur D. (Chart IV.) and George W. (Chart V.) are in point; in one the right apex and in the other the left apex was involved, but both were extremely mild cases, and in neither were there cerebral symptoms.

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[1] Emmett Holt, New York Medical Record, February 14, 1885.

The pulse and the respiration are very rapid in pneumonia of children, but this is by no means so unfavorable as it is in most other diseases. The pulse is frequently at one hundred and forty, one hundred and fifty, and even upward, in cases which recover, and, unless other and more unfavorable symptoms are present, we need feel no special alarm on this account. With regard to the respiration, it is common to observe a rate of forty to fifty in cases of only moderate severity, and it is rarely below thirty during the fastigium. When the rate rises above fifty, the chest should be carefully examined, to ascertain whether a large extent of the lung is involved, which would be an indication of danger; and it must be remembered that rapid breathing may be due to other causes than extensive hepatization; it may be caused by septicæmia, for instance.

A temperature above 105° F. if continued for several days is unfavorable, but when lasting only a single day, and especially when occurring suddenly and falling as quickly, is not necessarily dangerous: in fact, it often precedes the crisis (Charts V., VII., and VIII.). In some fatal cases it is very irregular, and it may fall a little shortly before death, as in the case of Harriet B. (Chart II.), where, indeed, the fatal issue was probably due to septicæmia rather than to the pneumonia. Sometimes in favorable cases the temperature will be extremely high at the outset and then fall immediately, as in the case of George D. (Chart VIII.) According to Thomas,[2] a want of correspondence between the range of temperature and that of the pulse is unfavorable, though high temperature with moderate pulse is less so than the opposite condition. Irregularity of the respiration, as well as of the pulse, is an indication of exhaustion, and consequently unfavorable. Termination of the fever by lysis instead of by crisis is not unfavorable.

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[1] Loc. cit., p. 65.

[2] Loc. cit., p. 707.

CHART VII.

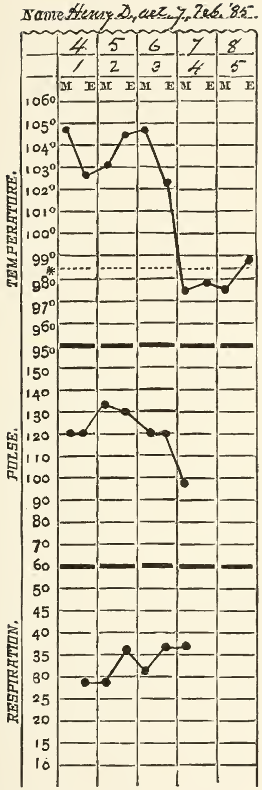

CHART VIII.

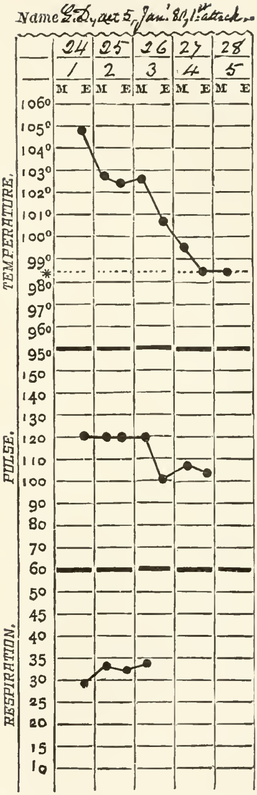

Chart VII.—Harry D., aged seven years. February 3, 1885, well and at school; restless at night; 4th, delirious, no cough, vomited; 5th, drowsy, slight loose cough, dulness and a few râles in lower left back, trace of albumen in urine; 6th, pain in left side, below nipple, cough, slight moan or grunt with each expiration; in the evening, bronchial respiration was heard at base of left back; 7th, he had a good night; this morning took milk for first time; sits up in bed.

Chart VIII.—George D., aged five years. "Slight cold" for a few days; January 24, 1880, pain in left side, cough and drowsiness, dulness and creaking râle in left lower back; 25th, dulness and bronchial respiration between angle of left scapula and spinal column; 26th, increased area of dulness, coarse râle in lower half of left back; at seven P.M., general improvement; 27th, rude respiration, no bronchial, and no râles in left back; took milk for first time; 28th, convalescent. (The only medicine given was a little Dover's powder.)

Francis Minot, M.D.

Delicate, cachectic children have comparatively little power of resistance to a disease which cripples the function of respiration, and almost all the fatal cases of primitive pneumonia are among that class of patients. In like manner, when pneumonia complicates the acute infectious diseases and other severe maladies of childhood, such as scarlatina, measles, diphtheria, septicæmia, tuberculosis, and general bronchitis, the prognosis is always doubtful, and usually grave. The supervention of a large pleuritic effusion would be a very unfavorable complication in a case of pneumonia, by further diminishing the area of breathing-surface, already much restricted, and also would suggest some fresh infection, as septicæmia.

Cerebral symptoms, as active delirium and mania, are alarming, but not dangerous except when indicative of meningitis,—a rare complication, in primitive pneumonia at least.

Treatment.—In the treatment of croupous pneumonia, as in that of all other diseases of children, the patient should occupy a sufficiently large and well-ventilated apartment, if possible with a southerly or southwestern exposure. The room should not be too much darkened, and the temperature should be moderate,—not exceeding 60° or 65° F. All noise and bustle should be interdicted, and the child should be disturbed as little as possible. If not much prostrated, his comfort will be promoted by a sponging daily, sometimes oftener, with tepid or warm water, but in cases of much exhaustion it is better merely to wipe the hands, feet, and neck with a wet rag.

Very little medicine is needed for healthy children in uncomplicated cases, and none should be given which is not clearly indicated. When the temperature is high, the skin hot and dry, and the patient restless, the compound tincture of ipecac (the "liquid Dover's powder") will be found very efficient. One drop of the tincture corresponds to one grain of the powder, and from one to three drops may be given to young children, to be repeated, when necessary, in an hour or two. The solution of the acetate of ammonium is also valuable for this purpose; it should be freshly and carefully prepared, and given in doses of from one to four drachms, according to the age, combined with equal parts of sweetened water, syrup of orange-peel, or other acceptable vehicle, several times a day, if required. For pain there is nothing equal to small doses of paregoric, five to twenty drops, repeated, if need be, in an hour or two. I have successfully treated several cases with paregoric alone, and some without even that. The tincture of aconite is often used in the treatment of pneumonia in children. I have frequently prescribed it, but I have never been able to satisfy myself that it is of any use, and, as it is a very depressing medicine when given in a full dose, it is not a convenient one to handle. From one-half of a drop to one drop of the tincture of the root may be given to a child two years old, when the temperature is high. Quinine is also frequently given for the sake of its refrigerant effect. For this purpose the dose must be so large as to cause much discomfort, at least, to the patient, and it is really, in my opinion, worthless in this disease.

Hot linseed poultices applied to the chest are employed by many physicians. It is difficult to see how they can act beneficially, since the object of treatment is to reduce the temperature rather than to raise it. They are, moreover, difficult to retain in place, and they offer an obstacle to the examination of the chest. I am satisfied, from my own experience, that in general patients are quite as comfortable and do quite as well without them, and I hardly ever employ them. If the patient is obliged to lie with the shoulders elevated, and therefore somewhat exposed, a light woollen jacket, or a waistcoat extemporized by cutting arm-holes in a piece of cotton wadding (not batting), will be found comfortable and convenient.

The bowels are to be attended to, but no purging should be allowed. The diet must be of the simplest kind. A little milk is all that most children will take until the temperature falls, and many will touch absolutely nothing but water. This usually alarms the friends, and sometimes the physician himself, who fears that unless the strength be supported by frequent administration of food the child will sink from exhaustion. There is no danger, however, from this abstinence. The digestion being arrested, no assimilation of nourishment can take place during the short period of the disease, but as soon as the crisis comes the patient demands food, which may then be given, at first in liquid form, and afterwards of more substantial nature. Plenty of water should be allowed throughout the disease, and lemonade or orange juice may be allowed.

If the child show signs of prostration, especially if the pulse should become feeble, irregular, or intermittent, stimulants are indicated, of which brandy is one of the best. Except in urgent cases, the amount required will be small. From five to twenty drops, according to the age, in a little milk, is usually sufficient, but sometimes much larger doses are necessary. Wine whey is an excellent substitute for brandy in mild cases, in doses of one or two tablespoonfuls.

The cough is rarely urgent enough to require special treatment, but, should it be troublesome, a few drops of paregoric are sufficient to moderate it. After convalescence has begun, if the cough is "tight," small doses of wine of ipecac are useful.

The convalescence from simple croupous pneumonia is usually uninterrupted and rapid, and in a few days the child returns to his usual diet, but the amount of solid food should be somewhat limited at first.

The cases of primitive pneumonia which do not follow a favorable course are chiefly those affecting delicate, feeble, or cachectic children, in whom the pulse is weak and rapid, the respiration shallow, the cough urgent but dry, and the skin of a dusky tint, the result of the ineffectual labor of the heart. Such patients require a sustaining and stimulating treatment, of which alcohol is an important part. Comparatively large doses of brandy are required, and from a teaspoonful to a tablespoonful, according to the age, may be given several times a day. Carbonate of ammonium is also a valuable remedy for this condition, and may be given

CHART IX.

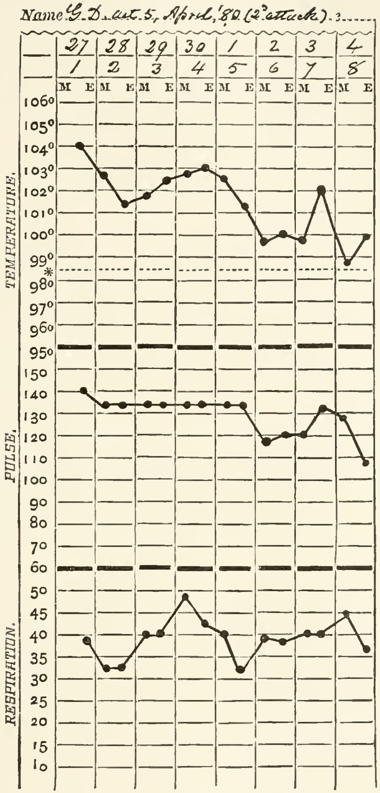

CHART IX.—George D., aged five years (second attack). "Took cold" April 23, 1880, but was about the house till noon of the 27th; went to bed at four P.M., with pain in the legs, frequent loose cough, hot skin, and vomiting; râle between left scapula and spinal column; 28th, vomited in the night, cheeks flushed, no bronchial respiration; 29th, herpetic vesicles on lips, dulness and bronchial respiration about angle of left scapula; vomited twice during the day; 30th, left cheek flushed, area of dulness and bronchial respiration includes lower two-thirds of left back; May 1, same condition and signs, cough looser; he has refused all nourishment since he was taken sick; 2d, took a little milk in the night for the first time, and some beef tea this morning; bronchial râle limited to spot two inches in diameter at angle of left scapula; 3d, good appetite.

Francis Minot, M.D.

BRONCHITIS.

By F. GORDON MORRILL, M.D.

ACUTE BRONCHITIS.

CERTAIN anatomical peculiarities of the child's lung deserve attention before entering upon a description of the acute affections to which it is most frequently subject. These peculiarities are of embryonic type, and are present to a greater or less extent up to the fifth year.

In the fœtus the bronchial tubes are relatively large, while the alveoli are mere bud-like dilatations, "as if nature had laid out a bronchial tree of generous proportions at the outset, to meet the demands of new-born existence and allow for its subsequent growth."[1]

The connective tissue in the fœtal lung is everywhere a delicate meshwork, but loosely retaining the blood-vessels, tending to abundant cell-proliferation, and occupying a far greater relative space than in the adult, —the air-vesicles and intervening connective tissue being about equal in extent.

The lining membrane of the bronchial tubes, with its rich net-work of capillaries, is but loosely bound to the muscular walls, and lies in folds. The alveolar walls are thick, and their structure comparatively loose and yielding; their inner surfaces readily shed and proliferate epithelium, as does the bronchial mucous membrane: moreover, the cells in both instances are relatively larger than those of an adult lung. The blood-vessels, being loosely restrained, readily dilate and encroach upon space properly belonging to the alveoli, and readily cause partial collapse. The thoracic walls are soft and yielding, while the muscles of the throat, bronchi, and chest are relatively less developed and far weaker than in the adult. These peculiarities (which, of course, gradually become less marked after birth as age advances) should be borne in mind, as showing the ease with which serious encroachment upon respiratory space may take place in certain inflammatory conditions.

By the fifth year, in a healthy child the loose connective tissue has become condensed, properly restraining the capillaries and binding the bronchial lining much more firmly to the walls. New alveoli have been produced, and the proper relative capacity of the air-spaces to the bronchial tubes has been established. The lung has now become anatomically adult, although it still preserves in a measure its faculty of easily shedding epithelium.

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[1] Dr. William P. Northrup, in Ref. Handbook Med. Sci., to whom I express my thanks for the free use which I have made of his paper.

Synonyme.—Acute bronchial catarrh.

Definition.—Acute inflammation of the whole or any portion of the lining membrane of the bronchial tubes, except that of the bronchioles,[1] which are probably never involved without implicating the air-vesicles,—in which case the disease can no longer be properly called bronchitis: it is broncho-pneumonia.[2]

An attempt to give a history of the disease would be to occupy space which can be better devoted to a more practical use. Suffice it to say that it was not accurately defined or its location fixed until after Laennec's discovery of the art of auscultation.

Etiology.—All mucous membranes are susceptible to congestion and inflammation from the effects of exposure to cold and damp. Cystitis and diarrhœa are familiar examples of this fact. But the relations between the bronchial lining and the skin are more intimate than those of any other mucous membrane. Moreover, certain anatomical peculiarities already referred to render children particularly susceptible to catarrhal inflammation.

Climate is naturally one of the most important factors in causation,—bronchitis being most prevalent in countries which are subject to frequent and sudden changes of humidity and temperature. That prolonged cold alone is a frequent cause is easily disproved by the rarity of its occurrence in the Arctic regions in winter. The New England States furnish an ideal climate for the production of this and other catarrhal troubles of the respiratory tract. In Boston bronchitis is most frequently observed during the early spring and autumn months.

Home surroundings and influences, including defective drainage and ventilation, deficient or excessive heating, insufficient nourishment, unsuitable clothing, and exposure to extremes of temperature, are prominent causes. Impure air, however produced, will irritate the respiratory mucous membranes, and bronchitis readily follows. In numerous instances a cracked furnace-pot has been proved responsible for attacks, repeated or prolonged, by inhalation of coal-gas.

The period of first dentition is one during which children are particularly liable to catarrhs of the respiratory tract. Jacobi lays stress upon the dampening of the clothing covering the chest by the free secretion of saliva in drooling babies. A neglected "head-cold" or slight laryngitis is often the starting-point of a bronchitis, and each attack weakens the mucous membrane and renders the child more susceptible to similar trouble. Certain diseases which alter the quality of the blood (notably measles[1] and whooping-cough) are accompanied with bronchitis.

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[1] The ultimate divisions of the bronchial tree from which air-cells begin to be given off.

[2] There are good authorities who believe that the bronchial epithelium (the character of which begins to change a short distance below the bifurcation of the trachea) ceases to be ciliated at a point which is at a perceptible distance from the ultimate divisions of the tubes. This would tend to strengthen the theory of the non-extension of the bronchitis into the bronchioles without involving the air-cells,—something more than a catarrhal process becoming necessary to carry the inflammation beyond a point where mucous glands cease and the epithelium, a single layer only, is "flat respiratory" like that of the air-cells.

One would naturally suppose that the sports indulged in by boys would render them more liable than the opposite sex to bronchitis; but this is not the case, so far as obtainable evidence goes to prove. Unknown atmospheric causes occasionally give rise to epidemics of the disease, and it may be safely said that the question "How can the child have taken cold?" is one far oftener asked than satisfactorily answered.

Pathology.—In its normal condition the bronchial lining membrane is bathed in its natural secretion to an extent sufficient for the normal performance of its functions. Inflammation produces a hypersecretion or catarrh which may involve the whole or any portion of the bronchial membrane where mucous glands are present. In severe cases the trachea is very frequently affected, and the disease is then one of direct and natural extension. When portions only of the bronchial tree are involved, the branches supplying the lower lobes are most frequently affected. During the acute stage the superficial columnar epithelium of the parts affected is shed and expectorated, very little being found present in the sputa of the later periods of the disease, during which cells of embryonic type are rapidly formed and shed, and together with the secretion of the mucous glands constitute the usual type of catarrhal products. In places where the inflammation has been severe, the color of the membrane varies from a pinkish red to deep scarlet, and the injection of the blood-vessels can be distinctly traced. The membrane is thickened, softened, and easily detached. In bronchitis accompanying measles, spots similar to those which constitute the eruption upon the skin have been observed. Besides these superficial appearances, in severe cases evidence of inflammation affecting the fibrous and muscular bronchial coats may be discovered, and the lymph-glands are enlarged.

Symptoms.—In children under five a catarrh of the nose and throat usually precedes bronchitis. The attack usually begins with a dry and somewhat harsh paroxysmal cough (due to the congestion and swelling of the mucous membrane), which is accompanied by a feeling of soreness in the trachea or behind the sternum. Similar sensations may be referred to the sides or epigastrium (by children old enough to make their feelings known) when muscular soreness from coughing supervenes. The inspiration is somewhat increased in frequency, but is painless. Nursing babies will frequently eject the nipple from the mouth and resume feeding after a short interval, the coryza accompanying the early stage materially obstructing their breathing-power. It would be extremely difficult, if not impossible, to express in figures the effect of bronchitis upon the rapidity of the pulse and respiration. Both are raised; but children differ so widely and vary so much in this respect that nothing approximating a definite rule can be formulated. Other things being equal, the more nervous the child the more rapid the action of the heart and lungs. The temperature seldom reaches 102.5° F. unless the alveoli have become involved and a far graver disease, broncho-pneumonia, is present. In cases of average severity 101° F. is rarely exceeded. The skin, as a rule, is dry, and the cheeks are flushed. There is no expectoration before the third or fourth day (or possibly until a week has passed), when in children over seven or eight a small quantity of viscid mucus is coughed up. In younger children it is usually swallowed, although I have more than once seen children of two and a half or three years expectorate perfectly well. Within forty-eight hours from the first appearance of expectoration the cough usually becomes loose and ceases to be paroxysmal, the expectoration is yellow and of thinner consistency, and in ten days or a fortnight from the commencement of the attack the disease usually disappears. Mild attacks may terminate in a week, while severe cases may last three weeks without deserving to be classified as "chronic."[1] A very slight streaking of the sputa with blood is occasionally observed, and has no special significance.

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[1] Strümpell (Text-Book of Medicine) considers the occurrence of bronchitis in many severe or prolonged cases of illness to be due to the inhalation of secretions of the mouth and pharynx containing bacteria, imperfect expectoration allowing mucus to remain and decompose in the bronchi.

Prognosis.—The prognosis of uncomplicated acute bronchitis in a previously healthy child with fair sanitary surroundings and proper care is favorable. Occurring in a feeble child,[2] or in one subjected to the neglect and unhealthful conditions which seem inseparable from poverty, it should always be guarded; for it is in this class of cases, in a vast majority of instances, that the bronchioles and alveoli become involved and broncho-pneumonia supervenes,—a statement which I hope to prove in discussing the last-named disease. The frequency with which "bronchitis" is credited as a cause of death among children in the annual mortuary statistics of large cities renders the correctness of the diagnosis open to doubt.

Diagnosis.—The comparatively painless cough, slight or moderate constitutional disturbance, and absence of dyspnœa and of physical signs pointing to grave diseases of the chest, render the diagnosis sufficiently easy in a great majority of cases. But in attacks of unusual severity, involving the smaller (never the smallest) tubes, and accompanied by marked constitutional disturbance, careful physical examination alone can enable us to distinguish between bronchitis and the other diseases with which it is liable to be confounded,—croupous pneumonia, broncho-pneumonia, pleurisy, laryngitis (which is one of its frequent accompaniments, but may of course be present independently), and the various forms of pulmonary phthisis. In uncomplicated bronchitis there is absence of dulness. Râles of various sorts and sizes (depending upon the stage of the disease and the size of the bronchi involved) are heard throughout or in some portion or portions of the lungs. Sibilant and sonorous râles are believed to be caused by swelling of the bronchial mucous membrane and consequent narrowing of the tubes, while moist or bubbling râles are attributed to the vibration of mucus. With the exception of the very fine crepitation which is pathognomonic of croupous pneumonia, every variety of pulmonary râle may be present. When sounds of the smaller sort are present in one lung only, the case should be regarded with suspicion and the diagnosis guarded until a sufficient time has elapsed to clear up all doubt as to the presence of tubercle or broncho-pneumonia: at the same time an uncomplicated bronchitis giving rise to signs in one lung only has been occasionally observed. Râles may frequently be felt by applying the palm of the hand to the back or side.

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[1] Should a bronchitis last for a longer period than this, I think it may be properly termed chronic.

[2] A slight bronchitis occurring in a child suffering from diarrhœa may cause pulmonary collapse and death. Dessau attributes bronchitis under these circumstances to pulmonary congestion, which he likens to the congestion in adults which often accompanies cholera. (Archives of Pediatrics, January, 1887.)

From the early stage of croupous pneumonia, bronchitis may be distinguished by its low temperature and absence of headache, vomiting, delirium, convulsions, and epigastric pain, some or all of which symptoms are present in a vast majority of cases of the former. In the more advanced stage of croupous pneumonia, dulness, dilatation of the nostrils, bronchial respiration, and in many instances the detection of fine crepitation render the two diseases easily distinguishable.

From a commencing broncho-pneumonia (often erroneously termed at this stage "capillary bronchitis"), or from a more advanced period in mild cases, the differential diagnosis is at times impossible; but a temperature of 102.5° to 103° F. together with any considerable degree of dyspnœa points strongly to an implication of the bronchioles and air-cells, although percussion-resonance is apparently normal. At the same time the symptoms just mentioned may be present without broncho-pneumonia developing, as illustrated in a case which I recently saw through the kindness of Dr. T. M. Rotch. In this instance the patient, a child of three, had coughed badly for forty-eight hours; the temperature (axillary) was 102.5° F., pulse one hundred and forty-five, respiration ninety. Numerous fine râles were heard throughout both lungs, and, notwithstanding the fact that no dulness could be detected, the case was regarded as one of developing broncho-pneumonia. The next day I was somewhat surprised at finding the child playing about the room, with a temperature of 100° F. and a respiration of thirty. Nothing had been prescribed besides a little brandy in milk; and I know of no way to account for the extremely rapid respiration unless it can be attributed to spasm of the bronchioles,[1] or a sudden congestion causing the blood-vessels to encroach upon space properly belonging to the air-cells. Certainly a temperature of 102.5° F. could not alone increase the rapidity of the breathing to such an extent. From marked cases or later stages of an average broncho-pneumonia the diagnosis is rendered sufficiently obvious by the absence of severe constitutional symptoms and physical signs which characterize the disease.

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[1] The bronchioles are completely surrounded by a muscular coat, the function of which is evidently to contract their diameter (a process easily effected in the smaller tubes, where cartilage is absent) and thus aid in the expulsion of mucus. According to Rindfleisch, this

In commencing pleurisy there is somewhat superficial respiration: the child evidently dreads taking a deep breath, and often shows by distortion of the face and frequent cries that it is in pain. The decubitus of children under three is usually dorsal, but in pleurisy they often lie upon one side,—*which*, depending upon whether greater relief is obtained from pressure or from free respiration.[1] As a rule, neither râles nor dulness can be detected in the earliest stage, but percussion of the affected side causes pain. In bronchitis respiration is painless, decubitus normal, and percussion may be freely practised without causing discomfort. Later on, signs of effusion are present in pleurisy, and at no stage of the disease is cough a prominent symptom; but when it is present the child tries hard to repress it, as it causes great distress.

In laryngitis there is hoarseness, while râles are absent. When this disease and bronchitis coexist, as frequently happens, and doubt exists as to which is the more responsible for the symptoms present, recourse to the laryngoscope may be had.

From phthisis caused by the cheesy degeneration of unabsorbed inflammatory products of either form of acute pneumonia, the previous history, emaciation, and persistent limitation of physical signs to certain portions of the lungs (usually the posterior middle or base) will enable us to make the distinction.[2] From incipient tubercular phthisis the diagnosis is difficult (often impossible) until after the disease has made some progress. A persistent hacking cough and continued elevation of evening temperature are suggestive of tubercle. Children as well as adults are often subject to cough from elongation of the uvula or the presence of follicular pharyngitis, either of which conditions is easily detected by an examination of the throat. As a rule, it may be said that children who vigorously resist physical examination are far more likely to have bronchitis than any of the more serious affections of the lungs.

Prophylaxis.—Less heat and more air are needed in children's nurseries, where an open fire should be used in cold weather and the temperature kept at 68° to 70° F. If a higher temperature is maintained, a bronchitis is the more easily contracted when the child goes out of doors. In young

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muscular coat can be traced as far as the alveolar passages, where it is reinforced and forms a sort of sphincter.

[1] Vogel, Diseases of Children.

[2] Even then the prognosis should be carefully guarded, for continued fever and the persistency of râles or even signs of consolidation which in adults would excite the greatest apprehension may disappear with surprising rapidity in children.

children the circulation is easily depressed, and greater caution should be exercised regarding exposure to cold and damp than in the case of adults. In very inclement weather children under five, unless perfectly robust, are safer in-doors than out. It is a good plan to open the windows of a room and allow a child (properly dressed) to play about in it and obtain fresh air without an unnecessary degree of exposure. Children too young or feeble to walk should be carried in the arms of the attendant while in the open air during cold weather. The clothing during the inclement seasons should be warm but light, and of a texture and shape which admit of free movement of the limbs and full expansion of the chest. A garment of loosely-woven fleecy cloth, lined with flannel, is warmer than one composed of a single thickness of stiff material which far exceeds it in weight. Moreover, a child thus clad can indulge in active exercise without the fatigue and perspiration which often follow slight exertion when dressed in stiff and heavy materials. Bathing the neck and chest in cold water the temperature of which has been gradually lowered from that of the morning bath is an excellent hardening process and can be pleasantly and safely carried out. A "head-cold" should be promptly checked ; and this can often be effected by means of a pretty thorough purge and a grain or more of Dover's powder. In case of a child who is subject to frequent attacks of bronchitis of mysterious origin, the house-drainage and the sanitary and heating apparatus should be carefully examined, and defects remedied if any are discovered. A rubber apron will protect the clothing from dampness when saliva flows from the mouth of a teething child. During any prolonged illness the mouth should be frequently cleansed and a mild disinfectant mouth-wash used.

Treatment.—Very mild attacks are sometimes apparently aborted, but I do not believe that the duration of an acute attack, after the disease is fairly established, can be materially shortened by the use of drugs. At the same time, much can be done for the patient's comfort, and there is no doubt that careful superintendence of the surroundings and the administration of certain remedies when clearly indicated contribute something towards a safe recovery and escape from the more serious troubles to which a bronchitis sometimes leads. In cases of any severity the child should be kept in a bed which should be so placed that avoidance of draught is secured. The temperature of the room should not exceed 70° F. In case of a robust child with a coated tongue a purge of calomel (gr. i–iii) may be administered with good results, while in those of more feeble constitution a few grains of rhubarb and soda answer the purpose sufficiently well. As an increase of tension is probably present in the bronchial arteries when a bronchitis is contracted, a purge (if not too severe) does good irrespective of the condition of the tongue. Possibly something may be achieved by the application of a mild counter-irritant (camphorated oil, for instance) to the chest-walls ; and in any event an outward application of some sort is always regarded with favor by the child's attendants. The feet should be soaked in mustard-water, and a few grains of Dover's powder given at night to check the annoyance of cough and promote action of the skin. Small doses of aconite in conjunction with sweet spirit of nitre are useful in reducing feverishness, and by dilating the small vessels arterial tension is lowered in the bronchi.

I think most authorities agree that by the above means mild cases of bronchitis are occasionally cut short or their subsequent course favorably modified, provided the treatment is carried out at the beginning of the attack. But opportunities of seeing bronchitis during the initial stage are comparatively rare. Usually the child is not seen by the medical attendant until after persistent cough has alarmed (or annoyed) the attendants, and bronchitis has become fairly established; and in discussing its proper treatment we enter debatable ground. A list of the drugs recommended and employed would include nearly every nauseant, depressant, and expectorant mentioned in the Pharmacopœia, and not a few whose names are never seen in print in this connection. Many of the doses prescribed are too powerful to be safely employed, while some of the mixtures are so disgusting to the taste as to render them a positive infliction to the child.

Expectorants are worse than useless during the dry stage, and should be strictly avoided until mucus is present in sufficient quantity to warrant their employment. Until this stage is reached, a few drops of the syrup or wine of ipecac every hour or two can do no harm, and are believed to hasten the advent of the second stage. They should always be given in a palatable vehicle. An opiate affords marked relief from the annoyance of the cough, particularly at night:

℞ Tinct. opii camph., ʒi–iv;
Syrup. tolutani, ℥i;
Aquæ, ad ℥iiss.
M.
Sig.—Shake, and take one teaspoonful.

The above can be used throughout the course of the disease, so long as night-cough is troublesome, the respiration easy, and no signs are observed of the blood not being perfectly well oxygenated. When the mucous flow has become fairly established, a mixture containing squill may be prescribed:

℞ Tinct. scillæ, ♏xx;
Syrup. tolutani,
Syrup. pruni Virgin., āā, ℥ss;
Aquæ, ad ℥iiss.
M.
Sig.—Shake, and take one teaspoonful.
For a child a year old.

Senega[1] and carbonate of ammonium[2] are useful in cases where a very stimulating expectorant is indicated by a feeble circulation, or the cough is not effective in ridding the lungs of the bronchial secretions:

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[1] Senega causes pretty continuous cough, either by stimulating the respiratory centre or by causing contraction of the muscular coat of the bronchi.

[2] Carbonate of ammonium should not be prescribed for young children in a proportion greater than one grain to the teaspoonful. In larger doses, if frequently given, it may produce acute gastritis.

℞ Ammonii carb., gr. v–x;
Tinct. scillæ, ♏xxv;
Syrup. senegæ, ℥ss;
Syrup. pruni Virgin., ℥iii.
M.

Sig.—Shake, and take one teaspoonful three or four times a day, as directed.
For a child a year old.

The proportions of the above mixture may be modified so that it can be administered at frequent intervals if it should be thought better to do so. It is not an agreeable dose, and is intended for use only in severe cases where expectoration (by which I mean, of course, in this connection the exit of mucus from the larynx) is wanting and collapse is feared. The muriate[1] may be substituted for the carbonate, in doses of two grains or more, if there is no indication for a cardiac stimulant.

Vomiting consists in a great measure of forced expiration attended by cough and evacuation of the air-passages as well as of the stomach; and in cases where mucus accumulates in the bronchi to such an extent as to cause dyspnœa, emetics, of which ipecac is perhaps the safest, are often very useful. Five to ten grains will usually produce fairly prolonged vomiting and retching without implicating the alimentary canal; but, unless a considerable quantity of mucus is got rid of and evident relief to the respiration follows, it is useless to repeat the dose if free emesis has been once obtained. Turpeth mineral is a speedy and sure emetic, and, although observation has proved that it may cause salivation and diarrhœa in adults, even after producing prompt and free emesis, I have never hesitated to give it in an emergency or in cases where ipecac has failed to act, and have thus far observed no bad effects from its use. With zinc, alum, or apomorphine I have had no experience in bronchitis, nor do I believe that the sudden expulsion of the contents of the stomach which the last-named drug produces can be particularly effective in ridding the bronchi of mucus. A more prolonged and continuous effort I should judge would be required, and I should hesitate to use it in case of a very young child, for fear of producing collapse.

Demulcents, like flaxseed and slippery elm, seem to contribute to the patient's comfort during the dry stage, and may be freely given. Should the excessive secretion of mucus continue after the acute stage has passed, a few drops of oil of turpentine in milk, or a mixture containing fluid extract of cubebs, may be tried, and usually some benefit will be derived. When the cough is spasmodic, as occasionally happens, terebin or eucalyptus oil should be given in small doses and the effect carefully watched.

Cod-liver oil and iron are extremely useful in debilitated cases where fever is not present to any extent and the stage of expectoration is prolonged. The former should be given in the form of a palatable emulsion, and the latter can be agreeably disguised as follows:

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[1] Alkalies stimulate the respiratory as well as the gastric tract.

℞ Ferri pyrophosphat., ℈i–ʒi;
Aquæ cinnamomi,
Syrup. simp., āā, ℥ii.
M.
Sig.—Teaspoonful three times a day.

The pyrophosphate is one of the most digestible of all ferruginous preparations, and I have never known a child to object to the prescription given above.

The diet should be easily digestible, and broths and soups substituted for meats (in cases of robust children who are old enough to eat it) during the acute stage. In younger children, whose habitual diet is composed of milk or some one of the infant foods, with occasional indulgence in more solid nutriment, the latter is best omitted until convalescence is established. In those who are naturally feeble, on the contrary, additional nourishment is required, and their strength should be sustained by stimulants and beef peptinoids throughout the entire course of the disease and during the period of convalescence.

A cup of warm broth or lemonade will be found to act as a pulmonary sedative in many cases, and enable the medical attendant to avoid in a certain measure the use of opiates. The mucus which children habitually swallow often produces abdominal pain and discomfort, which is readily cured by a laxative, which frequently brings away a large amount of the irritating material.

Antimony, which is a most potent cardio-vascular depressant, should never be prescribed for very young children, and whatever good effects it produces in older ones are obtainable by safer means. I am not prepared to admit that quinine "tightens a cough," but I have never observed the least benefit from its use in the bronchitis of children.

The following prescriptions have proved useful:

℞ Morphinæ sulphat., gr. ¼–i;
Chloroformi, ♏ii–x;
Syrup. pruni Virgin., ℥ii.
Sig.—Shake, and take one teaspoonful as directed.

This may be used to allay obstinate night-cough when milder remedies have failed. Its use must, of course, be guarded by explicit directions.

In cases where the mucous secretion continues for some days after the acute stage has passed, and the bronchitis threatens to become chronic, either of the following may be tried:

℞ Wine of tar (Wyeth's), ℥ii
Dose, fifteen drops to a half-teaspoonful four times a day.

℞ Ext. cubebæ fluid., ʒi–iii;
Chloroformi, ♏v–xii;
Syrup. pruni Virgin.,
Syrup. tolutani, āā, ℥iss.
M.
Sig.—Shake, and take one teaspoonful four times a day.

As a general rule, it is advisable in prescribing to separate medicines which are given to check the act of coughing from those which promote expectoration; but the following mixtures are often useful in cases where the bronchial mucus is tenacious and difficult to raise, or the cough during the "dry stage" is frequent and ineffective. Tolu and wild cherry, in the form of syrups, have probably very little therapeutic action. Whatever effect the latter may produce is very temporary, and its administration in small doses three or four times a day is practically useless. Both preparations, however, possess the rare merit of being agreeable to the taste; and young children seldom object to taking doses containing a sufficient quantity of either to disguise the flavor of remedies which given by themselves would be promptly declined after the first trial.

℞ Tinct. opii camphorat., ʒi–iii;
Ammonii chloridi, ʒss–ii;
Syrup. pruni Virgin., ℥i;
Syrup. tolutani, ad ℥iiss.
M.
Sig.—Shake, and take one teaspoonful p. r. n.

℞ Tinct. opii camphorat., ʒi–iii;
Syrup. ipecac., ʒss–ʒiiss;
Syrup. pruni Virgin., ℥i;
Syrup. tolutani, ad ℥iiss.
M.
Sig.—Shake, and take one teaspoonful p. r. n.

A large number of drugs of which no mention is made in this article are no doubt fully as effective as those which have been recommended. It is the writer's belief that confinement to the bed in a well-ventilated room which is kept at a proper temperature, together with a carefully-regulated diet, contributes more to the patient's recovery than any cough-mixtures, however artfully compounded, in a large majority of cases.

At the same time, something can be done to make a child (and its parents) more comfortable during an acute bronchitis by the judicious use of drugs. Mild cases can occasionally be aborted, the annoying cough safely controlled in a measure, and the hypersecretion of mucus after the acute stage is over is often promptly checked, by the means which have been described. Should they fail after a fair trial, a change of air will frequently bring about the desired improvement. Nor is it necessary to move the child to a distant point, as a rule,—any change, providing the air breathed is pure, usually proving beneficial. In conclusion, the writer would emphasize the fact that broncho-pneumonia (often disguised under the term "capillary bronchitis") *very rarely* supervenes in acute bronchitis of healthy children whose home surroundings are tolerably comfortable and cleanly, and that it is entirely unnecessary to keep the patients constantly on the verge of emesis with the idea of avoiding a danger which is purely imaginary in a vast majority of cases.

CHRONIC BRONCHITIS.

Chronic bronchitis in children is rarely seen in hospital wards, but is observed not infrequently in out-patient clinics and occasionally in private practice. There are two distinct types of this affection. The more common is that which consists essentially of a mere prolongation of the cough and expectoration which accompany an acute attack of bronchial catarrh. It is often difficult or impossible to account for this obstinate persistency of symptoms; but in many instances a rational explanation is afforded by the constitutional weakness of the patient, whose rallying-powers are defective. In other cases the degeneration of an acute attack into a chronic cough in an otherwise healthy child can be traced to injudicious exposure to cold or damp before thorough recovery has been attained, or to lack of proper and sufficient nourishment during convalescence. Any defects in heating, ventilation, or drainage, or of the manifold conditions called "sanitary surroundings," which allow the breathing of impure air, may be cited as causes of this form of chronic bronchitis. The symptoms are identical with those present during the stage of expectoration in acute attacks, and require no special notice. The patient may appear quite well in other respects, appetite, sleep, and strength may be perfectly good, but an obstinate cough and hypersecretion of bronchial mucus are present.

The other type is associated with signs of scrofula or of rachitis. The mucous membranes of scrofulous children are particularly sensitive and very subject to attacks of obstinate catarrhal inflammation, and the bronchitis in such cases is analogous to the chronic coryzas which are so common among this class. The influence of rachitis in the causation of chronic pulmonary catarrh is clearly recognized. Even without any deformity, the rachitical process is accompanied from an early period with bronchial and tracheal catarrh. A chronic cough in an infant, with very little or no fever, disappearing and returning, mostly with copious secretion,—which, however, is swallowed as soon as it reaches the pharynx,—rouses the suspicion of general rachitis.[1] Certain cases of convalescence from broncho-pneumonia and whooping-cough might perhaps properly be classified under the head of chronic bronchitis. Following whooping-cough an occasional recurrence of inspiratory spasm often tells the story of its origin; but, aside from this, "coqueluchoides" are liable to accompany chronic bronchitis in children who have never been subject to pertussis.

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[1] Jacobi, Pepper's System of Medicine, vol. ii.

Whatever the child's constitutional state may be, the physical signs are the same in all cases: coarse râles, dry, or moist, or both, heard throughout both lungs. If fine râles are present, they are usually confined to the lower lobes. The resonance is normal, unless emphysema is present. These signs, together with absence of fever, and a history of frequent cough with prolonged paroxysms morning and night, and, as a rule, copious expectoration, usually render the diagnosis sufficiently easy. That the cough is not due to a long uvula or a follicular pharyngitis can be proved by inspection. From fibroid phthisis, of which one occasionally meets with marked examples in children, the distinguishing features of chronic bronchitis are unimpaired resonance; diffusion of râles, which are not of the kind significant of consolidation or dilated bronchi; normal vocal resonance and fremitus; and absence of violent retching efforts, attended with profuse expectoration of purulent matter, which is often offensive to the smell, from retention in tubes which have lost their elasticity.

Prognosis.—Chronic bronchitis in children as compared with that of adults is benign and bearable. The chances of recovery in an otherwise healthy subject are infinitely better than in older people, for the reason that emphysema of any extent is rare, and if present may ultimately disappear, providing the bronchial lining resumes its normal condition. The state of the disease is very markedly influenced by atmospheric conditions; and after a succession of warm days the cough may entirely cease, but reappears in cold or damp weather. The supervention of other acute lung-diseases, in case the child's constitutional condition is otherwise satisfactory, is apparently no more to be dreaded in chronic bronchitis than in good health. The only exception to this rule is tubercle, of which a family history is occasionally obtained in children who cough for an indefinite time without impairment of their general health until this disease develops.[1] Occasionally collapse of a large area of lung occurs, which may prove fatal; but often reinflation takes place. I have more than once observed this, and have been surprised to see how slight were the rational symptoms accompanying unmistakable physical signs of atelectasis of a large portion of a lobe, as corroborated by prompt and complete re-expansion. In scrofula and rachitis, chronic bronchial catarrh is but one of a train of attendant evils, and is liable to develop at any time into a subacute broncho-pneumonia (rarely an acute attack) or to invite a deposition of miliary tubercle.

Treatment.—Benefit is often derived from the use of the class of remedies which includes turpentine, eucalyptus, copaiba, cubebs, and sandal-wood. Sugar and milk are the vehicles in which they can best be administered to young children, while older ones can often be taught to swallow small capsules. If a change of air can be had, it often produces a favorable effect in cases where drugs fail. Children whose health is impaired from any cause, including such as suffer from a constitutional taint of scrofula or rachitis, derive more benefit from cod-liver oil and iron (particularly the iodide) than from remedies addressed directly to the cough. The latter are frequently useful adjuvants in these cases, if given in a form which does not interfere with appetite and digestion.

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[1] J. Canby, Arch. Gén. de Méd., vol. ii., 1886.

In view of the liability of children suffering with chronic bronchitis to contract tubercle, they should not be admitted to hospital wards for treatment of this disease only.

MECHANICAL BRONCHITIS.

Mechanical bronchitis signifies an inflammation of the bronchial mucous membrane which is caused by the inhalation of any irritating substance sufficiently light to float in the air,—dust or minute particles of mineral or vegetable substances. In America it is rarely due to the employment of children in such industries as render workers liable to inhalations of this kind; and when it occurs it is usually caused by accidental and temporary exposure to an atmosphere favorable to its development, and promptly disappears when such exposure ceases.

PSEUDO-MEMBRANOUS BRONCHITIS.

Synonymes.—Plastic, Croupous, or Fibrinous bronchitis, Bronchial croup, Bronchial polypi.

Definition.—Bronchitis, acute or chronic, the distinguishing feature of which is membranous exudation of greater or less extent within the bronchial tubes.

Pseudo-membranous bronchitis is extremely rare at any age. Of seventy-six cases, eleven only occurred in children of twelve years or under. Its etiology is unknown. Neither tubercle, syphilis, rachitis, nor scrofula appears to be intimately connected with its causation. The disease may be diffused or circumscribed, and the period during which membrane is coughed up at intervals may vary from a day or two to months or years. Occasionally hæmoptysis precedes or accompanies the appearance of the bronchial casts. In seventeen cases occurring in children of twelve years or under, I find this symptom noted twice only. The shortest period during which membrane was thrown off was two days, and the longest four years.

Aside from the usual symptoms of bronchitis, certain signs are sometimes present which might lead to a suspicion of the true nature of the trouble before the appearance of casts makes the diagnosis positive. These are—absence of respiratory sounds, dulness over circumscribed areas,[1] dyspnœa, and dread of suffocation. The physical signs (when any are present) naturally lead to a suspicion of a pneumonia,—an error which is sometimes further confirmed by the casts being of such soft consistency and so striated with blood as closely to resemble rusty sputa. But the blood is superficial in the membranous expectoration, and is easily washed off, while in the sputa of pneumonia it is intimately mixed with the other elements. The presence of a foreign body in one of the bronchial tubes also gives rise to signs similar to those noted in some cases of membranous bronchitis, and in a vast majority of instances a correct diagnosis is not arrived at until the membrane is cast off and its nature recognized. Its expulsion is at times easy, and at other times accomplished only with the greatest effort and accompanied by suffocative paroxysms. Sometimes small pieces only are coughed up, while at other times complete casts of the bronchial tree, down to its minute ramifications, are cast off.[2] Great relief is experienced after getting rid of the membrane, which is white or yellowish white in color, and consists microscopically of a structureless fibrinous material containing leucocytes and occasionally, but rarely, red corpuscles and epithelial cells. The portions which come from the upper bronchi are recognized by their size and by the concentric layers of fibrin composing them.

Of the seventeen cases mentioned, the youngest was that of a child of three years, and four occurred in children of twelve. Seven were acute, nine were chronic, and the duration of one is not stated. Of the acute cases one was that of a boy, five occurred in girls, and in one the sex was not mentioned. The chronic cases were five boys and four girls. Of the acute cases four recovered. The result in a majority of the chronic cases is not given. According to Lebert, the disease is much more frequent (taking all cases, irrespective of age) in males than in females.

Treatment.—The treatment consists in the inhalation of alkaline steam (particularly lime-water, in which the membrane is said to be quite soluble), and the administration of iodide of potassium or mercury for their constitutional effects. The expectoration of the membrane when loose can be aided by emetics of ipecac and turpeth mineral. The strength should be supported throughout by easily digestible nourishment, and stimulants if required.

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[1] According to Riegel (Trousseau's Cyclopædia of Medicine), in cases of large bronchial casts firmly adherent, the inspiratory murmur is absent, but percussion-sonority is unimpaired. I have read two or three cases reported as occurring in children, where dulness on percussion was noted.

[2] A case of this kind occurring in a child of three was reported by Dr. Geo. H. Lyman, of Boston. (Boston Med. and Surg. Journal, 1878, vol. i. p. 108.)

CAPILLARY BRONCHITIS.

No term in the nomenclature of medicine has done so much to confuse the minds of students, and prevent men of experience from arriving at a common and definite understanding regarding important points in discussing the acute pulmonary diseases of childhood, as "capillary bronchitis." Within the past few years, however, its employment to describe a distinct and independent disease has diminished, and the space allotted to it in the writings of the best authorities has been abridged. It is seldom spoken of now as a bronchiolitis, but is generally described as "inflammation of the small but not the smallest bronchial tubes,"—in other words, a bronchitis which has reached the highest possible development without becoming a broncho-pneumonia,—a condition which cannot with certainty be recognized by either physical or rational signs; for even if broncho-pneumonia is present, its lesions may be too small or too deeply seated to afford evidence of their presence to the ear, while the rational symptoms may be utterly insufficient (even in undoubted cases) to base a pronounced opinion on, in the absence of more positive proof.

It has been repeatedly described as one of the most fatal diseases of childhood; yet accounts of autopsies are wanting where an inflammation of the small but not the smallest tubes has been verified by competent observers (after thorough microscopic search for evidence of other and more important pathological changes) as being the cause of death. Meanwhile, accounts of autopsies in cases where capillary bronchitis was credited with causing death, but where collapsed vesicles and other evidence of broncho-pneumonia, commencing or established, have been found post mortem, are common enough. Under these circumstances it would certainly appear wiser to avoid mention (at any rate, as a distinct and grave disease) of a condition which cannot be diagnosticated during life or verified after death.

The use of the term "capillary bronchitis" in other senses (to describe either the early stage of broncho-pneumonia or a condition in which the advent of this disease is merely feared or suspected), although perhaps not so objectionable as the one already alluded to, is nevertheless open to criticism.

The name has been so frequently associated with pulmonary collapse as to give rise to the idea in the minds of some that the two conditions are almost inseparable; whereas it is not uncommon to obtain perfectly good proof of collapse in feeble children, where implication of the small tubes by the insignificant amount of bronchial inflammation present is rendered extremely improbable by the speed (at times) with which reinflation of considerable areas takes place. Instances of this sort are by no means rare, and the very slight degree of disturbance of breathing which even pretty extensive collapse has been known to cause under such circumstances should be regarded as merely proving that a substitution of an entirely useless portion of lung in place of universally defective respiratory expansion has been effected, either by means of a mucous plug rendering a bronchus of some size impermeable to inspired air, or merely through a sudden loss of tension in the alveolar walls, neither of which conditions bears the slightest resemblance to "capillary bronchitis." Possibly "terminal bronchitis," if generally adopted in speaking of the utmost development which *per se* a bronchial catarrh is capable of attaining, would prove useful in doing away in this connection with a term which loose usage has made capable of conveying various meanings. This change, together with the invariable use of the word "bronchiolitis" to express inflammation of the terminal stems, which is essential to broncho-pneumonia, might after a time bring about the final disappearance of "capillary bronchitis," which has become a troublesome and intangible ghost, both in clinical teaching and in medical literature.

BRONCHO-PNEUMONIA.

By F. GORDON MORRILL, M.D.

ACUTE BRONCHO-PNEUMONIA.

Synonymes.—Acute lobular pneumonia, Acute catarrhal pneumonia, Capillary bronchitis,—and others which are obsolete.

Definition.—Acute inflammation of the bronchial lining membrane, which by direct extension and mechanical phenomena incidental to the disease involves the connective tissue, bronchioles, and air-cells. In severe cases every component element of the lung may become implicated by the inflammatory process, which assumes in each part the form proper to the tissue affected. The term "lobular pneumonia" is objectionable, inasmuch as it neither includes nor suggests the bronchial inflammation which is essential to the disease; moreover, an embolic pneumonia is anatomically "lobular," but is totally unlike the disease under consideration. "Catarrhal pneumonia" is incorrect, because other tissues than mucous membrane are involved to a marked degree. "Capillary bronchitis" is a term which admits of so many interpretations that its use, either to describe the early stage of a broncho-pneumonia or in any other sense, is to be avoided.

Prevalence and Mortality.—In our Northern cities broncho-pneumonia is very common among children under five years of age, and its mortality is large. The following table shows the total number of deaths certified as due to "bronchitis" and "pneumonia" among children under five, as set forth in the reports of the Boston Board of Health during a period of eight years,—1879 to 1886 inclusive. The whole number of deaths from the other most fatal diseases of childhood, during the same time and for the same age, is also given :

Bronchitis and pneumonia	2050	Croup	969
Cholera infantum	3577	Whooping-cough	624
Diphtheria	2180	Scarlet fever	624
Diarrhœa	1617	Measles	412

A vast majority of deaths credited to "bronchitis" were undoubtedly due to acute broncho-pneumonia, as one never hears of uncomplicated bronchitis being verified as a cause of death by competent observers of post-mortem appearances. That by far the greater number of those reported as due to "pneumonia" were caused by acute broncho-pneumonia becomes evident when we reflect upon the extreme rarity of fatal croupous pneumonia in young children, broncho-pneumonia being not only the more common but by far the more fatal disease among this class. When we also consider that the disease under consideration is one of the gravest complications of whooping-cough and measles, and may very well have been the fatal element in many of the deaths credited to them, we may safely estimate its direct fatality as equal to that of diphtheria and second only to that of cholera infantum among children under five years.

Again, it should be borne in mind that broncho-pneumonia is the starting-point of a large percentage of all cases of pulmonary consumption in children,—although very good authorities deny that this is the case unless a predisposition to tubercle is present.

Etiology.—Here, as in bronchitis, climate is an important element of causation. Sudden changes of temperature and humidity are the essential characteristics of climates in which the disease prevails.

The anatomical peculiarities of the child's lung described in the article on bronchitis should also be borne in mind. The relatively large size of the bronchial and respiratory epithelium, its irritability, the ease with which it is proliferated and shed, and the fact that inflammation in children is apt to be of embryonal type, are facts which require consideration in forming an opinion regarding the etiology of broncho-pneumonia.

It is during the period of dentition that the disease occurs with greatest frequency and is attended with greatest fatality. As the lung develops and begins to assume the adult type, it becomes far less subject to this form of inflammation, and the chances of recovery from the disease in case it should occur are greatly increased. In other words, *age* is a main factor of causation and fatality. The following table proves that a large majority of fatal cases occur during the first two years of life. I have included in it the deaths reported as due to "bronchitis," nearly all of which were undoubtedly caused by broncho-pneumonia.

Deaths from "Pneumonia" and "Bronchitis" occurring among Children in Boston during the nine years 1879–87.

YEAR.	UNDER 1 YEAR.		1 TO 2 YEARS.		2 TO 3 YEARS.		3 TO 4 YEARS.		4 TO 5 YEARS.		5 TO 10 YEARS.	
	P.	B.	P.	B.	P.	B.	P.	B.	P.	B.	P.	B.
1879	102	103	47	27	28	14	7	1	10	3	6	1
1880	114	160	61	44	32	9	16	6	18	5	10	2
1881	128	154	57	64	29	9	9	8	13	4	18	5
1882	78	179	44	54	17	20	15	7	11	1	17	14
1883	89	168	51	59	35	20	14	3	10	3	23	8
1884	91	204	62	65	24	21	7	8	9	4	24	4
1885	101	211	88	90	39	23	22	10	22	3	36	8
1886	117	191	57	53	26	42	17	8	4	2	19	9
1887	109	211	62	70	23	27	19	7	19	8	34	9
Total	929	1581	529	526	253	185	126	58	116	33	187	60

Again I wish to emphasize the fact that broncho-pneumonia is far commoner and infinitely more fatal than croupous pneumonia in children under five. That it is commoner most authorities agree. That it is more fatal can be proved by the records of autopsies performed in all large institutions where children are treated. The figures given above plainly show that the prognosis of pneumonia in children between the ages of five and ten, when the croupous form is the more frequent, is favorable as compared with that of cases occurring among those who are younger. The improbability of the mortality from bronchitis exceeding that of pneumonia, as would appear from the figures given (pneumonia 2140, bronchitis 2443), must be apparent to all. The table would lead us to infer that the differential diagnosis between the two diseases in children becomes easier as age increases, if we contrast the marked difference in the relative proportions of deaths from "bronchitis" and "pneumonia" before and after the third year.

One would naturally suppose that the more adventurous disposition of boys and the sports they indulge in would render acute pulmonary diseases more fatal to them than to the opposite sex. This theory is very slightly substantiated by the fact that of the deaths from pneumonia between the ages of three and ten, during eight years when the sex was obtainable, two hundred and fifty occurred in boys, and two hundred and forty-five in girls.

The influence of cold and damp can be readily seen in the next table, which shows the number of deaths from "pneumonia" and "bronchitis," in children under five, for each month during a period of six years,—1882 to 1887 inclusive.

MONTHS.	1882.		1883.		1884.		1885.		1886.		1887.		TOTAL.
	P.	B.	P.	B.	P.	B.	P.	B.	P.	B.	P.	B.	
January . .	17	30	18	26	29	32	31	46	18	34	32	38	351
February . .	14	24	13	22	21	33	30	33	18	28	17	35	288
March . . .	16	28	31	33	16	33	39	38	20	26	19	32	331
April. . . .	27	34	33	32	19	32	52	33	15	31	18	23	249
May	19	30	26	25	8	20	37	42	20	20	16	26	289
June	12	21	21	14	6	16	17	18	4	11	18	16	174
July	8	13	10	14	7	15	11	17	14	12	17	19	157
August . . .	4	9	5	16	8	10	9	11	9	11	15	13	120
September .	5	12	3	10	14	18	10	23	9	12	19	19	154
October . . .	10	8	9	13	10	14	10	22	15	20	14	34	179
November .	17	16	12	25	23	44	10	23	29	32	25	40	296
December . .	16	36	18	23	32	36	16	31	50	59	21	38	376

The comparative immunity from the fatal forms of acute lung-troubles during June, July, August, September, and October is clearly shown in the above table.

The bronchial inflammation which accompanies measles is extremely apt to involve the lung-tissues proper, and the influence of this disease upon the mortality from broncho-pneumonia is very marked. In 1884 and 1886 there were one thousand and two deaths reported from "pneumonia" and "bronchitis," and during these years one thousand and thirteen cases of measles occurred in Boston. In 1885 and 1887 there were eleven hundred and sixty-four deaths from the above-named diseases, while the number of cases of measles reported was four thousand and fifty-three. This increase of the mortality from the prevalence of measles can be demonstrated for almost any given period of time, irrespective of season. For instance, the aggregate mortality from acute pulmonary diseases during the summer months of 1882 and 1884, when Boston was comparatively free from measles, was one hundred and twenty-nine for children under five. During the corresponding months of 1885 and 1887, when measles were very prevalent, the returns showed a total of one hundred and eighty-one deaths from the above causes for children of the same age. The greatest mortality in any one month during four years, 1883 to 1886 inclusive, occurred in December, 1886.

Table showing the Mortality from "Pneumonia" and from "Bronchitis" during four successive Decembers, and the Number of Cases of Measles reported during these Months and the preceding Novembers.

YEAR.	PNEUMONIA.	BRONCHITIS.	TOTAL.	MEASLES.
1886	50	59	109	243
1885	16	31	47	30
1884	32	36	68	171
1883	18	23	41	0

That broncho-pneumonia is more prevalent among children whose sanitary environments are bad is a fact clearly recognized by more than one writer on the subject. Here in Boston it is a disease of the very poor, and, so far as its fatal form is concerned, is practically confined to this class. To verify this statement I have investigated the death-returns of three sections of the city, selected as representing so many distinct types of houses and occupants. Number 1 comprises the dwelling-portion of the Sixth Ward, which is Boston's poorest quarter,—overcrowded, and containing a large number of the dirtiest sort of tenement-houses. Number 2 is bounded by Washington and Tremont Streets and Chester and Union Parks. It comprises many fine private houses, more of medium cost, a large number of boarding-houses, and a few dirty tenements. Number 3 is bounded by Arlington, Beacon, Fairfield, and Boylston Streets. It contains a richer population and a greater number of costly private dwelling-houses than any other area of similar extent in the city. The streets are broad, and air and sunshine are plentiful. Neither section includes a hospital of any size. The three outlined blocks on the opposite page correctly show the comparative size of these sections, and a very close estimate of the population of each is given.

No. 1. Population, 17,000.

No. 2. Population, 5756.

No. 3. Population, 4600.

In 1884 the mortality among children under five in this city was: "pneumonia" 193, "bronchitis" 303. Section 1, pneumonia, 22, bronchitis 39; Section 2, pneumonia 3, bronchitis 0; Section 3, pneumonia 0, bronchitis 0. In 1887 the mortality for the city from above causes was: pneumonia 232, bronchitis 333. Section 1, pneumonia 23, bronchitis 33; Section 2, pneumonia 1, bronchitis 2; Section 3, pneumonia 1,[1] bronchitis 0. The above figures would seem to be fairly conclusive.

Another inference which might be drawn from these statistics is this. Although measles have been very prevalent during the past year (1887), and the mortality from the causes now under discussion raised in consequence of the fact, in Section 1 it was less than in 1884, when very few cases were reported. Consequently it would seem that among the poor who live in unhealthful sections the mortality is not influenced to any great extent by the presence or absence of measles. Of course nothing positive can be stated from a solitary observation like the above; but it suggests a question of some interest for future solution.

Lumbroso claims to have discovered the egg-shaped micrococcus (Friedländer's pneumococcus, observed also by Frobenius and Emmerich) in cases dying of broncho-pneumonia which followed measles, diphtheria, and croup; and he produced by its inoculation pneumonic inflammation in animals. Thaon[2] and Loefery both found bacilli in cases of fatal broncho-pneumonia following acute infectious diseases; but none tallied with Friedländer's description of what he considers the specific germ of croupous pneumonia, which is probably an infectious disease, and fastens upon the alveoli (as does typhoid fever on Peyer's patches) without affecting the bronchial lining or pulmonary connective tissue,—presenting a marked contrast in its morbid anatomy to the disease now under consideration. Again, we have no history of house-to-house infection of broncho-pneumonia, examples of which in the croupous form in pleasant weather have been pretty clearly shown by Flindt, at the International Medical Congress of 1884, and other competent observers. Undoubtedly a variety of bacilli have been observed in broncho-pneumonia, but that they may have entered the lung from the upper air-passages (where they are always present, as a result of decomposed secretions and food, in prolonged illness) is extremely probable.

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[1] An infant of three months; sick twenty-four hours; certificate signed by an irregular practitioner.

[2] Revue des Maladies de l'Enfance, February, 1886.

So far as can be judged from investigations upon this point up to the present time, it would seem probable that the disease is started by irritation of the bronchial mucous membrane from various causes, and is developed by direct extension and accidents incidental to the disease (as will be seen in the description of its pathology) to other tissues. When it occurs as a complication of measles, the initial (or bronchial) stage is often so short as to be unappreciable, so quickly are the bronchioles and alveoli involved. A case is cited by Northrup[1] which followed measles and terminated fatally in twenty hours. Notwithstanding its extremely short duration, pus escaped from the smaller bronchi, which were infiltrated and surrounded by congested zones. He remarks, "This may pass for a typical example of so-called capillary bronchitis; and yet there was beginning extensive pleurisy over both lungs, and unmistakable beginning pneumonitis." That the bronchial secretions absorb foul odors with great facility is shown by the result of an experiment by Richardson,[2] who, having a bronchitis, discovered that breathing in proximity to a jar containing decomposed brain of a sheep caused the expectoration to assume a fetid odor as readily as did the moist hand, moist cotton wool, or a watery solution of albumen. He was also able to verify the deodorizing influence of pure air by observing that the odor was no longer present in the expectoration after an out-of-door stroll. Children who are confined to the bed by prolonged illness are frequently subjects of the disease in a low, insidious form. Hypostatic congestion of the lungs and collection of bacteria in the mouth are both favorable[3] to the development of broncho-pneumonia of an insidious and chronic type. Children who are subject to scrofula and rickets are very prone to contract the disease. Occasionally, but rarely, it follows chronic bronchitis in an otherwise healthy child.

In view of the above facts, the conclusion is reached that climate, age, poverty, season, prevalence of measles, and impaired health from various causes are all important elements of causation. That a specific germ may yet be discovered is very probable; but there are other diseases (notably syphilis) whose demands for a revelation of the kind are infinitely more imperative.

Morbid Anatomy.—In a catarrhal inflammation of the bronchial lining membrane the cells of columnar epithelium, with their ciliated fringes which form the superficial inside coating of the tubes, desquamate and are replaced by cells of embryonal type, which are rapidly proliferated and shed. The débris thus formed, together with the secretion of the mucous glands, is coughed up past the glottis, provided the patient's powers of expulsion are equal to the task. In children the bronchial epithelium desquamates with extraordinary ease, and the proliferation of fresh cells is particularly active, while their expelling force, owing to deficient muscular development, is relatively slight as compared with that of adults. As the inflammatory process advances, the amount of bronchial secretion increases, and mucus is replaced by pus; and, although this change in consistency would render its expectoration easier if other things were equal, the increase in amount, and the impairment of the patient's naturally feeble expulsive powers by the duration of the illness, render it more difficult to get rid of. The two main branches of the bronchial tree naturally conduct portions of the secretion which the child is unable to force through the glottis to the lower and posterior parts of the lungs, in accordance with the sick child's natural decubitus; and it is these portions which are usually affected in broncho-pneumonia. The manner in which this retention of the secretion acts in assisting the development of the disease will be presently described. While the mucous membrane is pouring forth an abundant secretion, the inflammatory process *per se* is advancing irregularly in various directions,—not only to some of the bronchioles and air-cells, but *outward* to the bronchial walls and the surrounding connective tissue. "L'inflammation se propage par continuité."[1] Small round cells invade all the coats of a portion of the smaller bronchi. Sometimes a few only in a portion of a single lobe are thus involved; or the inflammation may be irregularly distributed and affect scattered groups, usually in the posterior portions of the lower lobes. Occasionally all or nearly all the smaller tubes in a single lobe are thus involved, resulting, together with the other pathological phenomena, in a condition sometimes called "lobar broncho-pneumonia." The advance of the inflammatory process may be extremely rapid and equivalent to an almost simultaneous invasion of all the tissues involved; or it may be slow and gradual, occupying weeks, or even months.

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[1] Ref. Hand-Book Med. Sci.

[2] Asclepiad, vol. iii., 1886.

[3] Strümpell calls this form "inhalation" or "deglutition" broncho-pneumonia.

A result of the infiltration of the bronchial coats in severe or prolonged cases is dilatation of some of the smaller tubes from loss of their elasticity; and in consequence of this there is a great diminution of their contractile power. These dilatations are generally fusiform, and situated, as one would naturally expect, in the lower lobes. The calibre of the smaller bronchi is greatly diminished by the swelling of their walls: "round nucleated cells known as embryonal cells appear, and tightly pack the loose tissue between the epithelial lining and the elastic bronchial walls."[2] Delafield lays great emphasis upon the fact that a zone of either intense congestion or genuine inflammation surrounds the smaller tubes, and that the morbid process can be distinctly seen to extend thence to the adjacent air-vesicles, the walls of which are attacked by this peri-bronchial pneumonia, and are infiltrated with cells exactly as are the bronchial walls,—showing a regular advance

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[1] F. Balzer, Nouveau Dictionnaire de Médecine, vol. xxviii.

[2] Northrup, loc. cit.

outward of the inflammatory process to the alveoli, without previous implication of the bronchioles or aspiration of morbid material being needed to achieve this result.[1] The connective tissue surrounding the bronchioles[2] is also inflamed, as well as that immediately surrounding the alveoli. Dr. J. M. Keating [3] lays stress upon this peri-alveolar inflammation as a probable factor in causing collapse by direct pressure. The interstitial character of many of the pathological phenomena probably accounts in a great measure for the severity and prolonged course of the disease. Meanwhile, the inner surfaces of the affected air-cells shed their epithelium and secrete pus. Moreover, a portion of the bronchial secretion which the patient is unable to cough up through the glottis finds its way into some of them. In addition to the above-described phenomena, there is intense congestion of the bronchial vessels, which materially aids in diminishing the lumen of the tubes, some of the smaller of which sooner or later become filled with secretion which has gravitated downward.[4]

This statement naturally leads to a description of collapse, how it is effected, and the important (even fatal) results which it may produce.

Collapse is an incident, or rather an accident, which invariably occurs to a greater or less extent during the course of a broncho-pneumonia of average duration and severity. I shall not stop to dwell upon the history of its discovery as a common pathological condition, and the various steps by which different investigators obtained the final and conclusive proofs of its true nature. Suffice it to say here that "collapse," "atelectasis," and the "fœtal state," when used in connection with broncho-pneumonia, mean one and the same thing,—*i.e.*, alveoli whose walls, when not prevented by a partial occupation of their cavities by the products of inflammation, are in apposition because they contain no air.

As already stated, a portion of the secretion natural to the disease finds its way downward instead of being coughed up and out through the glottis. To be able to appreciate properly the effects of this retention of inflammatory products, certain facts already alluded to should be borne in mind. In addition to the diminished calibre of the smaller tubes and the loss of contractility which is essential to the exercise of their expulsive powers, we have poorly-developed muscles (thoracic, bronchial, and laryngeal) weakened by illness. Under these conditions, mucus or pus readily finds its way to the bronchioles and alveolar passages, and the air-cells to which they lead collapse. The most ingenious explanation of the minutiæ of this phenomenon is that of Gaudner, who compares a small plug of mucus which has lodged in a bronchial tube to a ball-valve, which permits the escape but not the return of air, the aspiration of the partially-emptied vesicles drawing it farther and farther in, and the diminishing calibre of the tube bringing it in closer apposition with its walls at each respiratory act, while the expiratory acts and cough expel the remaining air, but cannot dislodge the plug. Finally, the air having become exhausted, the alveolar walls come in apposition, and atelectasis is achieved. Or, the vesicles having parted with the greater portion of their air, collapse occurs very slowly, the remaining air being gradually absorbed.

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[1] Phila. Medical News, November 15, 1884.

[2] The *ultimate* divisions of the bronchial tubes.

[3] Phila. Medical News, 1882.

[4] According to Branne and Stael (Archiv für Anatomie und Physiologie), when for any reason the capacity of a lung is diminished, the transverse section of the bronchus leading to it becomes less than that of a bronchus leading to a healthy lung. In other words, there is a definite and fixed relation between the lungs as containers and the bronchi as receivers of air.

The ball-valve theory certainly possesses the merit of great ingenuity, and may account for collapse during the early stages of the disease; but the character of the bronchial secretion changes materially as the inflammation progresses, and instead of viscid mucus pus is present: so that we must look for another cause of collapse occurring during the later periods of the disease.

Among the post-mortem appearances of broncho-pneumonia, the presence of pus in the smaller tubes is noteworthy in this connection. "From the finest bronchi can be expressed creamy pus containing air-bubbles,"[1] and it is to this pus in the bronchioles that collapse must frequently be ascribed. Expiration (proved by experiment to be one-third stronger than inspiration) and cough (which is expiration forced to its highest power, unless we except emesis) expel the air through the pus, which readily flows back if partially dislodged, or is aspirated back by the same vacuum which (aided by the outside pressure of poorly-restrained capillaries) draws the walls of the alveoli nearer together. It is extremely probable that pus is thus sucked into the alveoli; for when an aneurism bursts into the trachea, groups of vesicles are found to be distended with blood.[2] The microscope also affords evidence that in some instances the cellular elements present in the alveoli are derived from the lining membrane of the bronchi. By some authorities the migration of the bronchial secretions is regarded as an active agent in spreading the inflammation, a theory which finds pretty strong confirmation in the well-known irritating properties of the secretions of nasal and vaginal mucous membranes when inflamed.

We have, then, probably two distinct ways in which the inflammation may spread,—either by natural extension, or by the migration of bronchial secretions which act as irritants in places which the advance of the morbid process in the usual way has not reached. Vesicles may collapse without the inflammation reaching them by either of the above methods. Or they may become inflamed in either manner and then collapse, the portions of their cavities not occupied by inflammatory secretions parting with their remaining air in case their conducting bronchioles become obstructed.

To recapitulate: there is inflammation of the bronchial mucous membrane which involves the walls of the smaller tubes and the surrounding connective tissue by direct extension, and the bronchioles, alveolar passages, and air-cells either by direct extension or by the migration of inflammatory material. Moreover, this material may occasion collapse of groups of vesicles, an accident in the causation of which feeble respiratory power and narrowing of the lumen of the smaller tubes materially assist.

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[1] Northrup, loc. cit.

[2] Hamilton, London Practitioner, 1879–1881.

The post-mortem appearances which follow this complex pathological process vary in accordance with the severity and duration of the disease, and the predominance of inflammation or collapse. The presence of either of these conditions almost invariably implies the presence of the other, and both require a bronchitis of greater or less extent for their production. In some cases we find the pathological changes confined to scattered groups of the small tubes and their connecting alveoli,—"disseminated" broncho-pneumonia. In others a whole or nearly a whole lobe may be solidified by the aggregation of affected lobules,—"lobar" or "aggregate" broncho-pneumonia. Solidification may mean either inflammation or collapse, and the appearance of either of these conditions, when present to any extent, is usually sufficiently distinctive to enable the observer to form a rough estimate of their relative proportions; for, as already stated, both conditions are usually present. It may be said that collapsed lung is of a violet color, and its surface is shrunken, while inflamed (hepatized) lung has a raised surface, and its color is reddish brown. Another crude test is that of inflation, which certainly tells us something in cases where extensive collapse has recently occurred, inasmuch as freshly-collapsed air-cells can be distended by means of the blow-pipe, and the comparative extent of the atelectasis roughly estimated.

On the other hand, when collapse is of longer standing inflation may be impossible. Then, again, alveoli in which the inflammation is of recent date may be distended, their cavities being still permeable to sufficiently forcible air-pressure.[1] Northrup, whose statements are based upon the results of observations of between five and six hundred autopsies of children dying of pneumonia, says that he has "never failed to find in atelectatic areas abundant evidence of an inflammatory process both in the capillary bronchi and air-passages and in the alveoli," a statement which seems fairly conclusive. The microscope affords the only means of positively determining in doubtful cases whether inflammation or collapse is the true condition in certain areas, or whether both are present. Collapsed lung is of violet color, solid, non-crepitant, non-friable, and, being airless, sinks in water. This condition may be confined to scattered groups of vesicles, or may *apparently* be the only change present in an entire lobe. It is apt to be symmetrical in its distribution, and affects chiefly the posterior margins of both lower lobes, the lower margin of the middle lobe of the right lung, and often the lingula. On section dark blood escapes, and pus can be squeezed from the finest tubes. Separate areas of lung are often observed representing different stages of inflammation, from simple congestion to complete consolidation. Inflammatory consolidation, usually termed hepatization, is brownish red in color, and on section a thick reddish secretion can be scraped from its cut surface. At a later stage the color is about the same, but mottled by the presence of pus, and its cut surface yields on scraping a thick milky fluid. It is of firm consistency, but friable and frangible. Isolated groups of inflamed lobules are solid and firm to the touch, and often recognizable to the eye, for if superficial they are seen as small elevations above the surrounding surface. In size they vary from that of a small pea to that of a hazel-nut. By confluence of numerous hepatized lobules, large portions or even an entire lobe may become consolidated.

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[1] Bouchut (Maladies des Nouveaux-nés) stated, as long ago as 1869, that nothing very definite could be proved by inflation.

Under the microscope the alveolar walls are found to be lined with young germinal cells highly nucleated, and the lumen of the alveoli is filled with these and with mature epithelial cells in various stages of fatty degeneration, as shown by the presence of oil-globules.[1] This change in the character of the inflammatory product is favorable, constituting as it does an absorbable emulsion. Moisture tends to bring about this condition: hence the more acute the attack and the higher the blood-pressure, the better are the chances of speedy and complete recovery, as concerns this one result of a complex pathological process. Leucocytes, serum, and occasionally fibrin are found in the alveolar cavities, but the latter if present is seldom of any amount or of firm consistency. Blood is rarely observed. On the other hand, the products of inflammation may be found in a condition of commencing caseous degeneration, which is probably the starting-point of a large number of pulmonary phthises in young children. The inflammatory process in broncho-pneumonia is in a great measure essentially an exaggeration of nature's usual method of epithelial repair,[2] while that of the croupous form is an exudation of blood-solids into the alveoli.

Emphysema is frequently observed. It is usually vesicular, and if extensive chiefly affects the anterior surfaces of the upper lobes. The rational explanation of its presence is furnished by the diminished air-capacity of the portions of lung where broncho-pneumonia is present, and the forcing of air into the upper part of the chest during violent paroxysms of cough. The distended vesicles are plainly visible to the naked eye. It is thought that they return to their normal size upon recovery from the causative lesion. Some authorities believe that they rupture into one another; but the dilatations are generally of uniform size.

On the pleura patches of soft lymph are seen overlying the inflamed portions, and the membrane beneath them is found to be rough, congested, or ecchymosed. In places where the predominating lesion is collapse, ecchymosis is most common. Emphysematous blebs occasionally (but rarely) rupture into the pleural cavity, and produce pneumothorax. Well-marked exudations of lymph are sometimes seen, but anything like a free serous effusion is extremely rare, unless death occurs from a very severe type of the disease, such as sometimes prevails during an epidemic of measles, when serum or pus may be present in the pleural cavity. Small sub-pleural collections of the inflammatory secretions of the alveoli are occasionally observed.

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[1] D. I. Hamilton, op. cit.

[2] Hamilton, loc. cit.

In all inflammatory lesions of any extent the bronchial glands of children are swollen, and in broncho-pneumonia it is not uncommon to find in them evidence of the presence of miliary tubercle, even where it cannot elsewhere be discovered; or they may be enlarged to several times their normal size by simple hyperplasia. Acute miliary tuberculosis is not infrequently associated with the lesions of acute broncho-pneumonia. The liver and kidneys may be congested. Evidence of inflammation of the stomach and intestines is often observed, which may vary from a superficial catarrh to marked ulceration.[1]

Gangrene of the lung is such an extremely rare condition in association with acute broncho-pneumonia in children or adults, that I venture to insert here a description of the post-mortem appearances observed in a case of the kind which occurred in my service at the Children's Hospital. The disease followed measles, and the child, a girl aged three, had been ill during a period of eighteen days preceding the date of her admission to the wards. Autopsy by Dr. Wm. F. Whitney, twenty hours after death, which occurred next day after entering the hospital.

Rigor mortis absent. Livid discoloration of dependent parts of body. Moderate emaciation.

Head not opened.

Right side of *heart* filled with dark fluid blood. Perforation in the middle of the Eustachian valve three millimetres in diameter. Valves and muscular substance normal.

Left pleural cavity contained fifty cubic centimetres (by estimate) of light-colored serum. *Lung* not fully retracted; lower lobe of a dark-bluish color. Upon section of the upper lobe, the surface was found uniform, and considerable frothy fluid escaped upon pressure. The lower lobe was much more dense, of a uniform dark-bluish color, very moist, and here and there rather firmer nodules could be felt, from the midst of which a drop of muco-purulent fluid could be squeezed.

The *right lung* was firmly and extensively bound to the chest by comparatively recent adhesions, and covered in places by a layer of recent lymph. Numerous yellowish-white points could be seen through the pleura, and in one place this was necrosed. Upon section the upper lobe was found studded with numerous yellowish-white points and small nodules, intimately associated with the bronchi, the walls of which were somewhat thickened. In a few places these points had run together and small cavities had been formed. There were also several larger cavities formed from dilated bronchi. The lower lobe was of a dirty-reddish color; in the upper part was a discolored cavity crossed by shreds of tissue. The whole lobe was very much softened and highly offensive.

The *spleen* was slightly enlarged, firm, the trabeculæ and blood-vessels prominent. The capsule was thickened in places, and slightly adherent to the abdominal wall. The *kidneys* were normal in size; slightly pale in the cortical portions. The *liver* was large, doughy in consistence, of an opaque yellowish color; the outlines of the acini were not easily recognized. The *Peyer's patches* of the small intestines were marked out by a dark discoloration around the follicles, as were the follicles of the large intestine. The fæces were soft. *Lymph-glands* enlarged and cheesy.

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[1] William Pepper, Pepper's System of Medicine, vol. iii.

Diagnosis.—Acute broncho-pneumonia with gangrene of the lung, acute pleurisy, bronchiectasis, chronic peri-pleuritis, fatty infiltration of the liver, chronic intestinal catarrh.

Symptoms.—When acute broncho-pneumonia succeeds the eruption of measles (the disease in connection with which it occurs with greater frequency than with any other), it begins with the symptoms of an acute bronchitis. The inflammation advances rapidly and involves the pulmonary tissues. So quickly does this occur at times that it is impossible to define the stage during which the morbid process is confined to the bronchial tubes,—the bronchioles, connective tissue, and air-cells undoubtedly becoming involved before any proof of the fact can be obtained by physical signs of consolidation, which are seldom present before the third day. On the other hand, when the disease supervenes in cases of whooping-cough, its advent is slow and insidious, and is usually accompanied by a decrease of the paroxysms. Occurring as a distinct disease by itself, the length of time during which it may be preceded by acute bronchitis without any rational or physical signs of consolidation is extremely variable. In nine cases of this description the average duration of the bronchial stage was thirteen and a half days, the extremes being five and twenty-eight days. In three cases which followed chronic bronchitis only (so far as could be ascertained from the histories), cough preceded the signs of consolidation three hundred, two hundred and ten, and seventy-two days respectively.

The extension of the disease to the lung-tissue proper is accompanied with increase of fever, dyspnœa, and a change in the character of the cough, which becomes short, painful, hacking, and, as a rule, much more frequent. The respiration increases in frequency, and the working of the alæ nasi and anxious facial expression in cases of any severity show that the child's main object in life now is to obtain air. Its attention can only momentarily be distracted from its task (the importance of which it instinctively realizes) of obtaining sufficient oxygen to sustain life. Retraction of the ribs and intercostal spaces, chiefly in the lower and lateral portions of the chest, and depression of the epigastrium, are observed. The axillary temperature in the evening is found to be 103°–105° F., or even higher. The fever is of irregularly intermittent type, a variation of three degrees between the morning and evening temperature being common, and four degrees by no means rare. Occasionally the morning temperature may equal or even exceed that of the evening for a day or two. Vomiting and diarrhœa are frequently present during the acute stage, and the latter may continue throughout the entire course of the disease and far into the period of convalescence if the patient survives. I find one or both of these symptoms noted in thirteen out of twenty-nine cases in hospital practice. Expectoration is seldom observed in children under seven. When it occurs, it is never the typical sputa of lobar pneumonia, but consists of mucus or muco-pus, viscid or frothy, and occasionally, but rarely, streaked with blood. As fresh areas of lung become involved, paroxysms of dyspnœa occur and all the symptoms increase in severity. When areas of considerable size collapse, the dyspnœa increases, the temperature falls, cough may entirely cease, and evidence of carbonic-acid poisoning appears. The countenance is livid, the skin cool and moist to the touch, and, unless a radical change for the better takes place, death follows at an interval which rarely exceeds twenty-four hours. When successive portions of less extent collapse, symptoms resembling those just described appear, but their advent is more gradual, and the immediate cause of the unfavorable change is more difficult to recognize.

Or the patient may die of exhaustion from the prolonged fever and continuous struggle for breath, the disease pursuing a steadily unfavorable course and gradually reaching a fatal termination. When broncho-pneumonia follows the eruption of measles (and a majority of cases, I think, occur before the eruption has entirely faded), the duration is frequently short, and death or commencing convalescence may result within a week or ten days. But in a large majority of all cases improvement (when it takes place) is gradual, and the period of convalescence prolonged. The disease has no regular march of invasion. Its forces scatter and attack different areas of the lungs. The morbid process is complex, and absorption of the products of inflammation, as a rule, is so slow that it is extremely difficult to define the stage of resolution. Favorable symptoms aside from physical signs are decrease of cough and dyspnœa and a gradual assumption of a lower range of temperature.

The disease having been roughly outlined, special symptoms will now be considered.

Pulse and Respiration.—The frequency of the pulse is increased, and the rapid rate which it assumes during the early stages of the disease is apt to continue for a time after the temperature declines,—a fact which is accounted for by the weakness of the patient. In young children it varies from 120 to 200,—the latter number having been observed more than once in cases terminating favorably. This enormous variation is due not only to the degree of severity which the disease may assume, but also to the extremely variable pulse-rate in healthy children. As a general rule, it may be said that in cases of average severity the rate is 135 to 160 per minute. In the early stages it is full and tense, but as the disease progresses it becomes weak.

The respiration, like the pulse, is extremely variable, and the same remark which has been made regarding the normal pulse-rate of healthy children may be applied with greater force to the breathing. Vogel found the normal respiration of infants between three and four weeks of age to be 26.4 when asleep, but between 30 and 40 when awake. Excitement from any cause, however slight, at once produced a change of rate and rhythm. In children who have passed the infant stage the rate of respiration is still subject to great variations. When a broncho-pneumonia occurs as a complication of an eruptive disease, the frequency of the breathing is already increased by the high temperature natural to the accompanying fever. When it occurs independently, the respiration is but slightly raised during the stage when the inflammation is limited to the bronchi; but, as the bronchioles and parenchyma become implicated, the rate is at once increased, and sixty or seventy respirations per minute are often attained when the disease has become fairly established. Children suffer from dyspnœa more than adults with the same relative amount of lung involved. Slighter causes affect the frequency of their breathing: so that we must be prepared to encounter temporary periods of very rapid respiration, and to throw them out of our calculations in computing the actual rate in cases where they occur. The pain which accompanies the breathing in broncho-pneumonia can often be properly ascribed to the inflammation of the pleuræ; but when not due to that cause it is hard to account for it. To attribute it to the existence of the pulmonary lesions would seem hardly correct, when we reflect upon the slight amount of pain caused by actual necrosis of tissue in cases of pulmonary phthisis.

The rhythm of respiration is altered. The pause which naturally succeeds expiration now precedes it. Expiration is accompanied frequently by a moaning sound. The breathing of young children in broncho-pneumonia is no longer abdominal: the ribs rise and fall as in adults. Retraction of the intercostal spaces and ribs and depression of the epigastrium are caused by atmospheric pressure,—the lungs expanding incompletely, owing to a portion of their alveoli being rendered impermeable by inflammation or collapse. Dyspnœa is nature's response to the stimulation of the respiratory centre by deficiency of oxygen and presence of carbonic acid in the blood,—an effort to reverse the balance, which when prolonged tires out the respiratory muscles by shortening the resting-spaces and exhausts the vitality of the patient. Death in broncho-pneumonia results more frequently from respiratory than from heart failure. The advent of Cheyne-Stokes respiration is unfavorable, but cases in which it has been noted have been known to recover. Suffocative paroxysms occur in cases where collapse to any great extent, if present, cannot be detected by physical signs, and a fatal termination is occasionally averted by prompt and judicious treatment. Rilliet observed complete suspension of respiration lasting several minutes in an infant of two months, and left it, supposing death to have occurred. To his astonishment, he found it breathing a few hours later, and death did not take place until the next day.

Temperature.—An evening temperature of 104°–105° F. is common during the acute stage of severe cases. It may reach 107° F. and yet recovery follow. The highest temperature observed in twenty cases occurring in the Boston Children's Hospital was 106.5° F., and the result in this case was fatal. A remission of three or four degrees in the morning is quite usual, but the fever is very irregular, and it is not uncommon to observe a morning temperature which is considerably higher than that of evening,—

but this rarely continues for more than a day or two. There is no regular ratio between the pulse, the temperature, and the respiration. Chart I. is that of a very mild case in a child of fourteen months. The broncho-pneumonia was of a disseminated form, perfectly well marked, and followed by recovery.

In cases which accompany or follow measles, and in uncomplicated cases when considerable areas of lung are involved, the temperature is higher. Chart II. is that of a child aged seven, in whom broncho-pneumonia occurred during measles; death from exhaustion on the twenty-fifth day.

In cases of death from exhaustion the temperature often assumes a low range, and this is maintained for a period of some days previous to the fatal termination. A favorable result in broncho-pneumonia is never immediately preceded by an abrupt and extensive decline. This phenomenon when present means collapse, and is of the gravest import. Charts III. and IV. are examples of the temperature in death from extensive collapse, while Chart V. shows the favorable change in a case of croupous pneumonia by "crisis," and is presented as illustrating the diametrically opposed interpretations of similar thermometric incidents in the two diseases.

Occasionally the temperature immediately preceding death from collapse rises rapidly during the last few hours: 108° F. has been noted by more than one observer under these circumstances. Chart VI. is an example of a rise of five degrees accompanying a case of fatal collapse.

Croupous pneumonia sometimes reaches a favorable termination by a somewhat slow assumption of a normal range of temperature. But the prolongation of "crisis" is entirely unlike the tedious and irregular "lysis" of broncho-pneumonia. Chart VII. illustrates this statement.

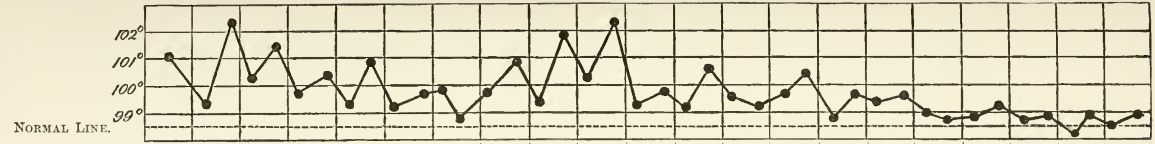
Digestive System.—The tongue during the early stage is usually coated, but may be red and irritable-looking. During the later stages of cases of any considerable duration, the mouth and tongue become dry, and sordes collect. Anorexia and thirst are present. Vomiting is not uncommon, but is rarely persistent. Diarrhœa of an obstinate character is not an unfrequent symptom, and is due to intestinal catarrh.

Nervous System.—Stupor, alone or alternating with delirium, is common in severe cases. At times the symptoms closely resemble those of tubercular meningitis; but when coma is present in the latter the temperature usually declines, while in broncho-pneumonia the range is maintained. In doubtful cases the ophthalmoscope is said to be of use at times in making a differential diagnosis.

Pleurisy is almost always plastic and circumscribed. It is rarely extensive enough to modify physical signs. Serous effusion is extremely rare. Purulent effusion has been observed by Pepper and Jürgensen. Pepper and Steffen have both noted the occurrence of pneumothorax. Gangrene of the lung in connection with broncho-pneumonia is extremely rare at any age.

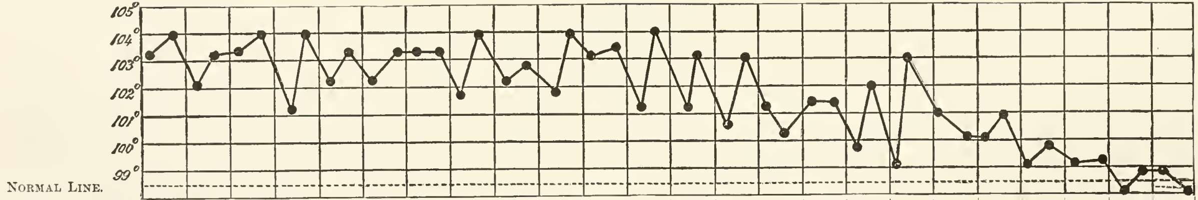
Diagnosis.—To distinguish between the early stage of the disease and

CHART I.



Pulse	A.M.	144	140	136	132	140	132	144	128	140	120	126	120	126	110	120	110	104	116	108	102	100
	P.M.	148	150	148	140	128	140	142	136	136	128	136	126	122	120	112	110	108	112	110	110	108
Respiration	A.M.	54	52	56	48	56	50	46	50	52	48	56	48	50	48	50	44	40	38	36	32	34
	P.M.	60	60	46	48	64	58	52	56	64	52	52	52	45	52	45	42	38	36	44	34	36

CHART II.



Pulse	A.M.	130	105	130	125	130	135	140	130	130	140	120	110	110	120	155	140	120	140	155	145	140	145	135	145
	P.M.	125	130	140	130	135	140	130	140	130	150	125	145	140	135	155	115	150	120	115	140	150	140	145	140
Respiration	A.M.				45	60	62	63	65	60	60	65	60	65	55	60	60	60	60	55	50	60	50	50	45
	P.M,			50	60	60	60	65	70	55	65	55	60	55	55	65	70	70	65	55	55	65	45	45	40

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an acute bronchitis is frequently impossible on account of the absence of signs of consolidation and the moderate degree of fever present. In cases where marked physical signs are wanting, but the rational symptoms are too serious to be accounted for by a bronchitis, the differential diagnosis usually lies between croupous pneumonia and broncho-pneumonia. The histories of these diseases differ materially, and, when a reliable account of the symptoms present during the commencement of the illness can be obtained, it is often a great aid in forming an opinion. In croupous pneumonia there may be vomiting, chills, pain in the epigastrium or abdomen, headache, delirium, or convulsions. It is extremely rare that all these symptoms are present in one case, but more than one of them may usually be noted by an attentive observer. In broncho-pneumonia there is often a history of measles, whooping-cough, scarlet fever, or bronchitis; and we very rarely obtain an account of apparently perfect health immediately preceding the attack, as is not seldom the case in the other disease. In croupous pneumonia the temperature frequently reaches 104°–105° F. within twenty-four hours after the first symptoms of illness are observed. In broncho-pneumonia the ascent is somewhat more gradual, and instead of a morning remission of two or three degrees, as is common in the croupous form, from three to five degrees is frequently noted. There is nothing absolutely distinctive in the pulse-respiration ratio; but in broncho-pneumonia it may be 1 to 2, or even 1 to 1.5,[1] while in croupous pneumonia the ratio oftener is 1 to 2.5. In croupous pneumonia the ratio is much more steadily maintained than in broncho-pneumonia, paroxysms of dyspnœa being common in the latter. Another distinction is that while in broncho-pneumonia the respiration is frequently laborious, in croupous pneumonia, while it may be equally rapid, the breathing is quiet (except for the expiratory moan common to both forms) and the accessory muscles are not brought into action. The patient's age is to be considered in connection with the diagnosis. It is during the dentitional period that broncho-pneumonia most frequently attacks children, while after this has passed either form may occur; but the chances are largely in favor of croupous pneumonia if the fifth year has been attained and the pneumonitis is the only disease present.

Nothing definite can be inferred from auscultation during the early stage. Râles and unimpaired resonance are common to both forms of pneumonia in children. Occasionally the limitation of dry râles to one apex leads us to suspect that croupous pneumonia is being developed; but the moist râles which are so common in broncho-pneumonia throughout the back are also frequently heard in the same location in the other form. It is only where the disease has made a certain amount of progress that the physical signs are sufficiently distinctive to enable one to make a positive diagnosis; nor can this be done at all in a large number of cases, without considering the rational signs, and carefully weighing all the evidence obtainable from the history, appearance, temperature, and respiration of the patient, in connection with that which examination of the chest affords.

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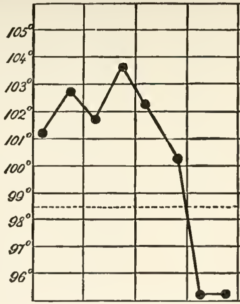
[1] I have never observed a ratio expressed by the latter figures except in cases where symptoms of extensive collapse were present.

At the same time the physical signs of well-marked cases of the two forms differ materially, and careful auscultation and percussion yield good results when the disease is fairly established. In broncho-pneumonia evidence of consolidation is usually obtainable in both lungs before the case ends, while in croupous pneumonia in a vast majority of instances it is confined to one lung. Of one hundred and ninety-one cases, in six and three-tenths per cent. only was evidence of consolidation in both lungs obtained. Again, the upper lobes are much more frequently affected in the croupous form (twenty out of fifty-one cases observed by Meigs and Pepper) than in broncho-pneumonia. In either form nervous symptoms predominate in case the apex is involved. In croupous pneumonia it is usually easy to demonstrate quite an area of dulness (which may be partly due to an accompanying pleurisy) at a comparatively early stage of the disease, while at a corresponding period in broncho-pneumonia percussion may show nothing, either because the consolidation is deep-seated, or because there is no aggregation of affected lobules sufficiently large to modify the percussion-note. Usually, however, a lack of resonance (possibly more appreciable to the touch than to the ear) can be discovered in disseminated cases, while in the "aggregate" form dulness may be present on an entire lobe. In cases where extensive collapse is present, dulness may be found along the spinal column on both sides. The dulness caused by broncho-pneumonia comes and goes slowly, so far as my experience teaches, and I have never seen the sudden changes from it to comparative resonance so graphically described when percussion has been carefully practised during both inspiration and expiration and the child has remained quiet. Another common error is that of mistaking normal hepatic for lung dulness; and a diagnosis of pneumonia (either form) based on the fact that slight dulness can be detected over the posterior base of the right lung in a child where nothing more significant than moist râles can be heard is always open to suspicion. On auscultation during the early stages of broncho-pneumonia râles of all sorts and sizes may be heard, but at a later period there are persistent subcrepitant râles in one or more spots. Well-marked bronchial respiration over consolidation is rare; in its place there are very apt to be weak and blowing breath-sounds. In croupous pneumonia, on the other hand, bronchial respiration is common, and there is often a fine crepitant râle audible on the edges of the consolidation, together with bronchophony and well-marked increase of vocal fremitus. Both the last-named symptoms are practically absent in broncho-pneumonia, and over collapsed areas of any extent inspiration and vocal fremitus may be entirely wanting, in recognition of which fact it has been quaintly said that care should be exercised lest one locate the pneumonia where one hears the most noise.

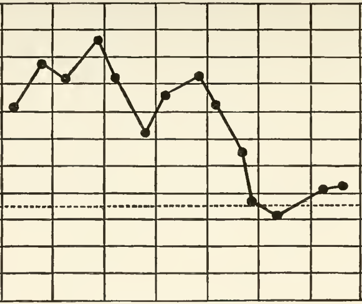
The duration and modes of termination in favorable cases differ materi-

CHART III.



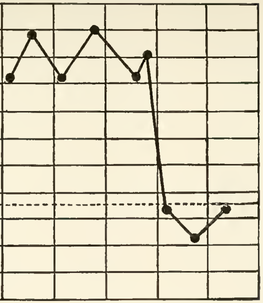
A. B., aged two and one-half years.

CHART IV.



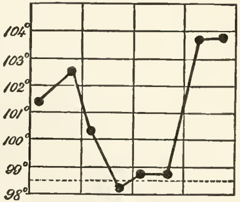
C. B., aged three years.

CHART V.



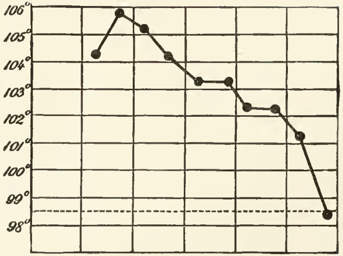
C. M., aged seven years.

CHART VI.



A. S., aged thirteen months.

CHART VII.



F. C., aged seven years.

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ally in the two diseases. In croupous pneumonia convalescence (preceded by crisis) is usually established in a week or ten days, while in broncho-pneumonia it is attained by lysis, and the duration of an active pathological process is much longer in a vast majority of cases which recover.

Broncho-Pneumonia.	Croupous Pneumonia.
Child usually under three years of age.	Child usually over three years of age.[1]
Often immediately preceded by measles, scarlet fever, or whooping-cough.	Usually immediately preceded by good health.
Usually, at the most, chilly sensations only.	Attack begins with one or more of the following symptoms: chills, headache, pain in epigastrium or abdomen, delirium or convulsions, and vomiting.
Temperature not so high, and rise more gradual.. Remissions of three to four degrees very common. Fever very irregular.	Sudden rise of temperature, which may reach 105° F. within twenty-four hours. Remissions averaging two degrees. Fever tolerably regular.
Breathing rapid and laborious. Accessory muscles of respiration used. Paroxysms of dyspnœa.	Breathing rapid, but not laborious, and accessory muscles of respiration not called into play. No paroxysmal dyspnœa.
Pulse-respiration ratio 1 to 2 or 1 to 2.5.	Pulse-respiration ratio 1 to 2.5.
Consolidation of greater or less extent in both sides. Râles heard over both lungs. Apex very rarely involved.	Consolidation one-sided and not infrequently involving an upper lobe. Râles occasionally heard in both sides.
Dulness seldom extensive; usually in more than one spot. Very often merely a sense of resistance on percussion.	Marked dulness over a considerable area not infrequent.
Moist subcrepitant râles, persistent in spots. No fine crepitant râles, but occasionally coarse crepitation. Over spots where dulness is detected the respiration is apt to be weak, absent, or blowing.	Fine crepitant râles on the edges of consolidation, and bronchial respiration, bronchophony, and increased vocal resonance over centre. None of these symptoms necessarily present, but all may be.
Duration indefinite, but much longer than that of the croupous form in a vast majority of cases which recover.	Duration from onset to sudden drop of temperature preceding convalescence seven to ten days.
Lysis.	Terminates by crisis in cases which recover.
Often leaves permanent lesions. Very fatal.	Recovery almost always perfect. Recovery the rule.

The symptoms, both rational and physical, of broncho-pneumonia differ essentially from those of pleurisy. During the early stage of pleurisy the pain in the side or abdomen, slight alteration of the pulse-respiration ratio, and maintenance of strength contrast strongly with the early symptoms of broncho-pneumonia. In pleurisy, percussion over the affected spot often produces pain, which is seldom the case in broncho-pneumonia. After effusion is present, the percussion-flatness, which can be found *in front* as well as over the back of the chest, absence of râles, and bronchial respiration which is often heard at or just above the level of the effusion are very different from the scattered and ill-defined dulness (usually confined to the back), subcrepitant râles, and weak or blowing respiration which are common in broncho-pneumonias of any extent. Again, although a pleurisy may be double, it is one-sided in a very large percentage of cases, while broncho-pneumonia, as a rule, affects both lungs. In cases of large effusion there may be obliteration of the intercostal spaces of the affected side, which is actually increased in measurement, and one may get total extinction of voice and respiratory sounds, or ægophony, and displacement of the heart. In pleurisy a change of the child's position may produce marked alteration in the sounds heard on percussion and auscultation. In conclusion, it may be safely said that the painful and superficial breathing, the suddenness of the attack, and the one-sided symptoms of pleurisy are far more often mistaken for croupous pneumonia than for broncho-pneumonia.

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[1] In seventeen typical cases treated at the Children's Hospital, the average age was four and six-tenths years, average morning remission of temperature about two degrees, and the duration of the disease up to the occurrence of the crisis between eight and nine days.

Prognosis.—The prognosis should always be carefully guarded, even when small areas only are apparently involved. It should be borne in mind that deep-seated consolidation may exist which cannot be detected, and that the invasion of successive areas is common, so that what may seem a light case, so far as physical signs are concerned, may actually be a severe one, or is liable to become so very shortly. As a rule, the younger the child the fewer are the chances of recovery. Meinssen lost fifty per cent. of his cases under one year, forty per cent. under three years, and twenty-five per cent. over three years. Bouchut lost thirty-three out of fifty-five cases under two years. When broncho-pneumonia follows measles it is usually of extremely acute type, and, although very fatal, recovery if attained is more apt to be complete than in the more protracted form, which oftener results in permanent lesions. Authorities differ widely in their estimates of the mortality of acute broncho-pneumonia, nor will it seem strange that they should do so if we reflect upon the various pathological conditions with which it is associated. In three hundred and twenty-five cases which I have collected from various sources (the only qualification necessary to be included in this number being that of age,—*i.e.*, under ten), the mortality was forty-eight and three-tenths per cent. Following whooping-cough the disease is of an obstinate and fatal type.

Special symptoms which are of the most importance in estimating the chances of recovery in a given case are—the extent of lung involved, as shown by the physical signs, temperature and pulse-respiration ratio, collapse, and amount of resistance of which the child is capable, as shown by its previous health and present general condition. Other things being equal, the more extensive the inflammatory process, as shown by the physical signs, the greater the danger of an unfavorable result. A temperature of over 105° F., if maintained for any length of time, is very unfavorable. A pulse-respiration ratio of one to one and one-half usually signifies that death is near. Collapse, if accompanied by a marked decline of temperature and great lividity of the countenance, is the gravest of all incidents which are liable to happen during the course of an acute broncho-pneumonia, and death is usually the direct result unless a decided rally takes place within twenty-four hours. But the child's powers of resistance may be so great as to carry it safely through extensive consolidation, a temperature of 107° F., and grave signs of collapse; or they may be so slight that it succumbs to a broncho-pneumonia of less than average severity. A rachitic child, or one whose health is in any way impaired, is of course less likely to weather the storm than one whose health is good when attacked.

But, whatever the child's history may be, there are various things to be considered about its present condition in estimating its powers of resistance. Is nourishment taken, and is it retained? Is diarrhœa present, and, if so, how serious is it? Is the pulse weak and irregular, and, if so, how do stimulants affect it? Does the child take notice at all? Is it delirious? Is any portion of the bronchial secretion coughed up clear of the glottis? By close attention to points of this kind we are sometimes justified in believing that there may be a chance of recovery, however desperate the case may appear to be. In cases where broncho-pneumonia kills by its continued fever and tiring out the muscles of respiration, the temperature often assumes a low range for some days before death: so that symptom should be regarded as ominous unless accompanied by improvement in the pulse and respiration. However light a case may appear to be, it is never safe to prophesy complete recovery. On the other hand, that complete recovery may be attained, even after weeks or perhaps months have elapsed without any improvement in the physical signs, is a well-known fact.

Prophylaxis.—The abolition of the two chief underlying causes of the disease, measles and bad hygienic surroundings of the poorest class, is a task which health authorities are trying hard to achieve, and in which medical men can materially assist. It is only within a few years that the gravity of measles has come to be properly appreciated, and even to-day one occasionally hears of parents foolishly congratulating themselves upon the fact that their children have contracted the disease, "because they can all get through with it together." At such times as educational and domestic affairs unite in furnishing a favorable opportunity for children to be sick, ignorant people have been known to expose them voluntarily to the infection. I need not say that it is the duty of every physician to combat vigorously such absurd ideas and censure careless or wilful exposure.

The immediate prophylaxis of broncho-pneumonia consists in the prevention or effective treatment of catarrhal troubles of the respiratory tract, and the careful cleansing of the nose, mouth, and throat during the course of any protracted illness. The use of a pleasant mouth-wash (to be applied with a swab if necessary) is also indicated:

℞ Listerin,
Glycerini, āā, ℥ss;
Aquæ, ℥iv.
M.

The above is usually acceptable to children, and possesses some antiseptic properties.

Treatment.—The disease cannot be cut short by any means known at the present time. To nourish the child, make it as comfortable as possible, and promptly use appropriate remedies in such emergencies as may arise, should be the chief aim of treatment.

The patient should be placed in a good-sized room where there are ample facilities for admitting light and air. The temperature should be maintained at 68° to 70° F., and, if at any time it is necessary to open a window to reduce it to this point, no fear need be entertained of so doing, provided the child is not exposed to draughts. An open fire should be kept burning. These recommendations, of course, apply mainly to the rare cases of children in good circumstances who have contracted broncho-pneumonia. As a rule, it is in hospitals and tenement-houses that we encounter the disease; but even in the latter something may usually be achieved towards improving the air which the patient breathes. A light jacket, composed of an outer layer of cotton cloth, an inside layer of cotton flannel, and an intermediate one of batting, should be worn. The lower halves of the "arm-sizes" should be cut out in the usual way, and secured over the shoulders with loops of tape, and across the front of the chest with small safety-pins. It should be carefully adjusted in such a way as not to fit too closely for free respiration, nor loosely enough to allow it to become crumpled or rolled up.

Outward applications should be avoided as useless unless emergencies arise which call for their employment. The time-honored flaxseed poultice should not be countenanced in this connection. By oiling the chest it prevents perspiration, and its weight is an additional tax upon the respiratory muscles, to say nothing of the trouble involved in its preparation and application.[1] The jacket just described is more cleanly, answers all purposes equally well, and is far more convenient as regards examinations of the chest. Blisters,[2] irritating ointments, and strong mustard pastes are unnecessary and harmful.

Little attentions which increase the child's comfort should be promptly but not fussily bestowed. The pillow and bedclothes should be rearranged and straightened, the mouth and lips kept clean and moist, and the child bathed every day without removing the coverings. In short, it should be well nursed.

During the first stage the harassing cough (and consequent loss of rest) is the chief active source of discomfort, and is to be treated with opiates of strength appropriate to the age and condition of the patient. The same remedies that have been recommended to check the troublesome cough in cases of bronchitis are applicable in broncho-pneumonia. But when the disease is fairly established the greatest caution should be exercised in the administration of opiates, lest the sensibility of the respiratory centre become blunted, and the reflex cough which rids the bronchi of obstructing mucus cease. I am aware that good authorities entirely oppose the exhibition of opium during any stage of the disease; but I believe that, if properly used and its effects carefully watched, during the early stages, it is often a safe means of obtaining refreshing sleep for the child, and saving its strength, which will shortly be taxed to its utmost.

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[1] I once witnessed the death of a child which was directly caused by too hot a flaxseed poultice applied by an apparently intelligent nurse.

[2] Gangrene has been known to follow the application of strong mustard as well as blisters.

Ipecac is given early in the disease in routine practice, for the reason that it is believed to hasten the secretion of the bronchial mucous glands and in some way benefit the patient. It is extremely doubtful if stimulating the mucous glands can perceptibly influence so complex a morbid process as that of broncho-pneumonia. Moreover, to keep a child on the verge of emesis (as many of the doses recommended must inevitably do) whose strength is already impaired (and will shortly be more so, by the struggle to obtain air enough to support life) cannot be good practice. Again, why should there be any anxiety on the part of the medical attendant to hasten or increase the advent of mucus and pus in the bronchial tubes? The mucous glands will soon pour forth an abundance of catarrhal secretion, and very likely there will be enough to cause collapse and endanger life, without any assistance by artificial stimulation. Ipecac should be reserved to use in emetic doses, in case it is indicated at a later stage, to rid the lungs of the catarrhal product, which often accumulates to such an extent as to obstruct respiration. Its exhibition in any other way is more than useless when broncho-pneumonia is present.

During the first stage of the disease a mercurial purge is often indicated, in children of average physique, by a coated tongue. Small doses of calomel with bicarbonate of sodium answer the purpose:

℞ Hydrarg. chlorid. mit., gr. i–ii;
Sodii bicarb., gr. xxiv.
M. Ft. chart. no. xii.
Sig.—One powder every other hour until a movement is obtained.

In cases of feeble children two or three only of the above powders should be given, and the bowels then moved by an enema if necessary.

The diet should be easily assimilable and at the same time as nourishing as possible,—milk, broths, and cereal food preparations,—and in severe cases stimulants should be given from the beginning. All treatment by drugs should be made subordinate to the nourishment of the child. White of egg stirred in cold water with the addition of a little sugar and brandy can often be given and retained in teaspoonful doses when everything else is either positively declined by the patient or rejected by the stomach. Children take brandy better than any other stimulant. Champagne, which is usually so grateful to sick adults, is seldom relished by young children.

Jacobi approves of the free use of water in cases of broncho-pneumonia in children, on the ground that it helps to induce fatty degeneration and absorption of the inflammatory products. This recommendation is practically carried out by adhering to the diet mentioned above.

After the disease is fairly established, it should be remembered that *cough* is the child's chief means of defence against pulmonary collapse, and the use of opiates in cases of any severity is contra-indicated, while stimulants, both alcoholic and expectorant, are often imperatively called for. In such cases, when the acts of coughing are infrequent, or ineffective (as shown by dyspnœa and the rattling of mucus which the child fails to force through the glottis), the following is a useful combination:

℞ Ammonii carb., gr. v–x;
Tinct. scillæ, ♏xx–xxx;
Syrup. senegæ, ʒii–iv;
Syrup. pruni Virgin., ℥ii.
M.
S.—Shake, and take a teaspoonful every two hours.

The strength should be supported by brandy, which should be given in as large quantities as the patient can bear with comfort; and the amount which children can take without being flushed or sleepy is at times phenomenal.

Should the symptoms continue in spite of our efforts to enable the child to expectorate by inducing cough[1] and increasing its strength, emesis should be promptly induced, with the hope that in this manner the lungs may be freed of their obstructing mucus. For this purpose ipecac in five-grain doses may be used, and is generally effective. Turpeth mineral in doses of two or three grains (repeated in ten minutes if not effective) usually acts promptly, and should be given without hesitation if less powerful emetics fail. For further consideration regarding the employment of this and other emetics for the purpose of clearing the bronchial tubes, the reader is referred to the article on acute bronchitis. Should no considerable amount of mucus be expelled by emesis, it is not advisable to reduce the patient's strength by further efforts in this direction.

In case the dyspnœa is paroxysmal, and not evidently caused by obstruction of the bronchial tubes, the introduction of steam to the child's bedside is often serviceable in softening the viscid mucus (which may not be sufficient in quantity to cause actual obstruction) and acting as a pulmonary sedative. This is easily done by moving the bed to a corner of the room and arranging sheets over it in such a way as to make a tent; the steam may be generated from teakettles, or more conveniently by hot bricks in pans of water placed around the bed beneath the sheets.

Should this method fail, or for any reason be inconvenient, the child should be stripped and gently submerged in a bath of a temperature of

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[1] Senega usually induces pretty continuous cough.

100° F. If very young, it can be placed in a large towel gathered at the head and feet like a hammock, and gradually lowered into the water until all but the face is covered. This manœuvre, if carefully executed, seldom excites any apprehension on the part of the patient, who should be taken out at the expiration of ten or fifteen minutes, wrapped at once in a blanket, and laid on the bed, where it frequently gets a refreshing sleep. The same means may be employed (several times a day if necessary) to reduce a high temperature, which may be in itself a cause of dyspnœa as well as a menace to life. Under these circumstances the temperature of the bath can be gradually lowered to 80° F., and a little brandy administered while the child is submerged.

Cold applications are recommended by many authorities, but, aside from the fact that they often disturb and frighten the child, the probability that equally good results may be obtained in a way more agreeable to the patient (and, I may add, to the attendants) renders their employment unadvisable unless other means have failed and the continued elevation of temperature is regarded as dangerous.[1] Both antipyrin and antifebrin in doses appropriate to the age of the child are effective in reducing temperature, and often contribute to the patient's comfort. Quinine in doses sufficient to effect any marked change in this respect is regarded by many good authorities as a more potent heart-depressor than aconite; while in moderate quantities it produces no effect whatever, so far as I have been able to observe.

The effects of any and all remedies which can be safely used to reduce the temperature of broncho-pneumonia are temporary, and repetition at longer or shorter intervals is required to produce noticeable benefit. An important consideration before employing any means for effecting a reduction is this: children differ widely in their constitutional capacities for bearing fever-heat. A temperature of 103° F. will often give rise in one child to symptoms of dyspnœa and distress which would be entirely wanting in another with a temperature of 105° F. or even more. The treatment should be with reference to the condition of the child,—not to the condition of the thermometer. Collapse, which manifests its presence by cessation of cough while râles remain, livid countenance, rapid respiration, and low temperature, requires bold and energetic treatment. The hypodermic use of brandy or sulphuric ether, the application of mustard cloths to the chest and legs, the alternate hot and cold douche,—any and all means of rousing the child and exciting cough should be promptly resorted to and persevered in so long as life remains. Mouth-to-mouth inflation has been used with success in cases of collapse. During the course of the disease the child's position should be frequently changed, to prevent hypostatic congestion and œdema and thereby render the occurrence of collapse less likely. After the purge administered (if thought advisable) at the commencement of the illness, the bowels should be relieved by enemas, as diarrhœa may set in at any period of the disease, and its advent might be hastened, or perhaps caused, by laxatives.

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[1] Dr. Loomis (Text-Book of Prac. Med.) advises strongly against their use.

As the child seems better and the temperature and pulse assume a lower range, if the hypersecretion of mucus continues, oil of turpentine, wine of tar, or cubebs may be tried, and one or the other will generally be found beneficial. At a more advanced stage of convalescence, cod-liver oil and iron are of the greatest service in renewing the child's strength and hastening the absorption of obstinate deposits in the lungs. After a siege of broncho-pneumonia the patient's spirits are usually subdued, and there is seldom any difficulty experienced in getting it to take its oil pure, and its iron in the form of syrup of the iodide. Should it object, the oil may be given in the form of a palatable emulsion (or in capsules to older children), and Rabuteau's syrup may be substituted for the plainer preparation of iron. Another most excellent tonic, which children take well, is Parrish's "chemical food" in teaspoonful doses. It has been used extensively, and with good results, at the Boston Children's Hospital during the past twelve years. The syrups of the lactophosphate and pyrophosphate of iron are also eligible preparations. Alcoholic stimulants, if used during the acute stage, should be continued, and, if not employed earlier, will be found very beneficial in shortening the period of convalescence.

Counter-irritation with dry cups, or by painting with tincture of iodine (which should be cautiously employed, lest a bad dermatitis be produced), and forced inspirations, are useful in promoting absorption. If circumstances permit, a change of air will often aid materially in bringing about perfect recovery. Great caution should be exercised regarding exposure to cold and damp for a long time after apparent recovery has taken place.

SUBACUTE AND CHRONIC BRONCHO-PNEUMONIA.

Incidental reference has already been made to a low form of broncho-pneumonia which is liable to supervene during the course of any prolonged illness involving confinement to the bed. The disease in this connection is of subacute and insidious type. Rational symptoms may be wanting to such an extent as to render the discovery of quite extensive consolidation purely accidental. As a rule, however, the knowledge of a child's liability to broncho-pneumonia as a complication of such troubles as necessitate the maintenance of a recumbent position for a considerable space of time leads to frequent examinations of the chest, and the discovery of the pulmonary trouble as soon as physical signs of any extent are present. Depressed vitality, hypostatic congestion, and the gravitation of bronchial secretions containing bacteria generated in the mouth and throat from sordes and decomposing food are the factors in this slow development of a subacute inflammatory process which does not essentially differ in its minute pathological features from an acute broncho-pneumonia.

The gross post-mortem appearances are included by the term "carnification," which differs from "hepatization" only inasmuch as it describes the results of a slow instead of an acute inflammatory process. Its disposition is often symmetrical, as it is prone to involve the posterior margins of both lungs, especially the lower lobes. Collapse probably plays a more important part here than in the acute form, as might be expected from deficient respiratory expansion through weakness caused by the illness which originally caused the child to become bedridden, prolonged gravitation of fluid and hypostatic congestion of the pulmonary vessels supplying the inferior lobes causing encroachment upon space properly belonging to the air-cells. Carnified lung varies in color from dark violet to dark mahogany. The violet hue, which resembles that of atelectasis, is due to prolonged congestion. Its cut surface is smooth, uniformly level, and not granular. It is not frangible nor easily torn. On inflation air may be made to enter slowly and irregularly into such portions as are not fully solidified. Microscopically, evidence is readily obtained of the presence of the same process (differing only in degree) which obtains in more rapidly developed cases of broncho-pneumonia. The prognosis of this form of the disease (occurring as it does in subjects already weakened by prolonged sickness) is extremely grave.

The chances of its occurrence may be materially lessened by frequently changing the child's position and keeping the mouth and throat clear of sordes and alimentary débris. The use of detergent mouth-washes is also strongly indicated. So far as treatment is concerned, the supervention of a broncho-pneumonia during the course of any illness is merely an indication for still further exertions to support the child's strength by every known means.

Subacute broncho-pneumonia may also occur as an independent disease, in which case it usually attacks a single lung and affects the apex oftener than the base. It may occur in children who are free from any predisposition, inherited or acquired, to organic lung-troubles. In a respectable percentage of cases the history, physical aspect, complete recovery, and subsequent good health of the patients would confirm the truth of this statement.

On the other hand, a subacute broncho-pneumonia oftener fastens upon those whose inherited or acquired physical traits render them particularly susceptible to "genuine" tubercular deposits, or their practical equivalents in the form of caseous degeneration of inflammatory products; and there can be no doubt regarding the very essential relations which broncho-pneumonia bears to the development of pulmonary phthisis in children. No theory can hold good which is based upon a belief that portions at least of the caseous masses in the lungs are not substantially identical with the products which are so characteristic of pulmonary inflammation in children where the air-cells are secondarily affected. Absorption of these products is slow, even in the most favorable cases; and an attempt to disassociate them from the cheesy deposits found where fatal pulmonary consumption directly follows either an acute or a subacute attack of broncho-pneumonia is to imply a duality of physical vitalism, which both promotes the absorption of a material requiring an increase of strength on the part of the patient before this process can be accomplished, and favors the substitution of another material whose deposition certainly cannot be interpreted as a sign of returning health.

The changes which take place as results of the presence and degeneration of the inflammatory material of broncho-pneumonia are essential features in a vast majority of cases of ulcerative destruction of the lung in children, and are mentioned here only for the purpose of emphasizing the inseparable relations of the two diseases, without reference to the part played by the tubercle-bacillus, or to the diagnosis and treatment of pulmonary consumption,—the discussion of which has been assigned to most able hands. When a broncho-pneumonia of any degree terminates favorably, fatty degeneration of its inflammatory products precedes absorption in a very large majority of cases. This favorable change in the character of the deposits may be indefinitely delayed, may be arrested after being begun, or may entirely default. Under any of these conditions nature may effect a cure by a deposition of earthy salts and the formation of a fibrous capsule, provided the lesion is small. But this is a rare occurrence.

As a rule, cheesy degeneration sooner or later takes place in deposits which obstinately resist the benign advent of oil-globules (or suffer them to pine through lack of companionship), the alveolar walls break down, and cavities form. In other instances these phenomena are overshadowed in importance by persistency of inflammation in the bronchial walls and inter-alveolar septa, which results in a dense formation of fibrous tissue, the shrinkage of which produces a diminution in size of the lung, and dilatation of the bronchi. The lesions which follow a subacute broncho-pneumonia are usually seated at the apex, while those which remain after an acute attack are oftener found at the base. Cheesy masses are frequently discovered in the bronchial glands, the centres of which are sometimes broken down and contain a creamy fluid.

"Genuine" tubercle may precede the inflammation in the lung, or may be subsequently deposited.

EMPHYSEMA.

By FREDERICK C. SHATTUCK, M.D.

The term emphysema implies an excessive quantity of air in the lung,—a disproportion between the air and the solid tissue in favor of the former.

Vesicular emphysema is that form in which the air is still contained within the air-vesicles, which are in a condition the precise opposite of atelectasis. Of vesicular emphysema there are two subvarieties: 1. The term "substantive" is applied to that form in which the disease is of apparently independent origin,—primary and general rather than secondary and local, permanent rather than temporary. 2. "Vicarious" or "compensatory" emphysema, on the other hand, denotes that form in which certain portions of the lung or lungs are overdistended with air as a consequence of a diminution in the amount of air contained in other portions, of greater or less extent, for this or that cause. This form is, therefore, always secondary.

Interstitial emphysema signifies the presence of air within the tissues themselves, either of the lung, pleura, mediastinum, or subcutaneous fatty layer.

Etiology.—1. Substantive or primary emphysema is, in children, a rare disease: indeed, apart from the emphysema which may be found in otherwise healthy lungs and which dates simply from the death-struggle, it is very rare and almost confined to the last years of childhood. James Jackson, Jr., first suggested that emphysema may be inherited. The supposition that nutritive changes in the lungs, especially in the elastic tissue, play a very important part in the production of emphysema is worth mentioning here, it being quite conceivable that an abnormal delicacy of the elastic fibres should be transmitted from parent to child. Gerhardt thinks that heredity may well help to explain those cases in which the affection develops at the time of second dentition.

Hecker's remarkable case shows that emphysema may arise during birth, from premature attempts to breathe. It may also be produced by efforts to resuscitate still-born children by means of blowing air into the lungs. Some medico-legal interest attaches to the question whether air found in the lungs of a new-born infant may be a product of decomposition. It is hardly conceivable that such a change can take place soon after death. These possibilities, as well as emphysema dependent on the death-agony, are of more purely scientific than clinical importance.

2. Secondary or vicarious emphysema, greater or less in degree, is, on the other hand, very common in childhood, and may arise from a large variety of causes which all have the common factor of interference with the easy and thorough performance of the respiratory act. It would lead us too far to attempt to enumerate all the special forms of such interference. The most important come under the main heads of mechanical obstruction in the upper air-passages; cessation of function in considerable portions of one or both lungs, either from consolidation or from compression, thus involving extra work on the part of the sound portions; and prolonged or violent cough,—forced expiration against a closed glottis,—the result of which is increased expiratory pressure, the accessory muscles being called upon for aid. Thus, in practice, the two most common causes for this form of emphysema are whooping-cough and bronchitis, especially when the smaller tubes are involved and atelectasis or lobular pneumonia follows. While some of the chief causes of vicarious emphysema are thus markedly frequent in children, and while the lungs of the young are probably more easily overdistended than are those of adults, these causes are generally of comparatively short duration and the reparative processes are more active during this period of life. Hence it results that on the cessation of the cause the effect usually disappears more or less promptly. Severe pertussis, which probably always brings about some degree of emphysema, is far more common than marked emphysema in later years for which a cause other than the whooping-cough can reasonably be assigned. At the same time it cannot positively be denied that in some cases the beginnings of an adult emphysema may date from a whooping-cough, bronchitis, catarrhal pneumonia, or other more or less acute affection which was apparently fully recovered from in early life.

Among the causes which may produce a permanent emphysema in children may be mentioned obstruction to the trachea by goitre,—rare in this country; tuberculosis; adhesive pleurisy localized about the margins of the lower portions of the lungs and preventing free retraction in expiration; and interstitial pneumonia starting either from the pleura or from the connective tissue of the lung itself. In the two latter cases bronchiectasis may be associated with the emphysema. In general it may be stated that the permanency of the emphysema depends in the first place on the length of duration or permanency of the cause, and in the second place on the presence or absence of an hereditary weakness of the elastic tissue. Much has been written in the past about the relative importance of the parts played by inspiration and expiration in producing emphysema. Either may be operative, but the latter is preponderant. Finally, the disturbances of the circulation in the lungs due to the persistence of the fœtal channels or a defective septum are associated with emphysema, which Gerhardt thinks, arising in the first days of life, may be causative of the cardiac defect. Rachitic deformities of the chest and vertebral column may also lead to emphysema.

3. The etiology of interstitial emphysema can be more briefly dealt with. Under the greatly-increased pressure of violent cough the delicate alveolar walls may rupture, and allow the escape of air, first into the interlobular, later perhaps into the subpleural tissue. If the opening is small or soon closed, the escaped air is readily absorbed. But if the air continues to pass out, it may find its way along the trachea or sheath of the vessels into the subcutaneous cellular tissue. The alveolar rupture may be due to external injury or violence, or to forced respiration into the air-passages of an asphyxiated infant.

Pathology.—The pathological anatomy of emphysema in childhood differs essentially from that in adults. Fürst, indeed, is inclined to doubt the existence of a genuine emphysema, recognizable microscopically as well as with the naked eye, in children. The air-vesicles are distended, but their partition-walls are not ruptured or atrophied, there is no notable destruction of the capillaries of the alveoli, and the elasticity of the lungs is not so much impaired that its loss is evident in hardened sections. There can be no question that Fürst is correct as regards the vast majority of cases, and we can thus understand better the frequency with which the process is entirely recovered from. Real tissue-changes are absent. At the same time the gross appearances are marked enough. On opening the body of a child dead of a pulmonary affection, one is apt to be struck by the increased volume of the lungs, which do not collapse as do healthy ones when air is admitted to the chest. The pericardium may be nearly or quite hidden by the distended left lung, and the diaphragm and abdominal organs lying beneath it depressed. The anterior surfaces of the lungs may be indented by the ribs, and raised at points corresponding to the interspaces. The emphysematous portions are pale in color, and the pigmentation along the interlobular tissue is ordinarily much less marked than in adults. These changes are not usually uniformly distributed, but are localized especially at the apices, and along the anterior and inferior edges, which are more rounded than in health. When the affected portions are incised, the air escapes without the fine crepitation characteristic of the normal lung.

To turn now to the rare cases in which a genuine emphysema develops in children, the cause being permanent. Here we may have the same series of changes as occur in grown people,—coalescence of the alveoli, atrophy and loss of elasticity in their walls, destruction of capillaries, heightened pressure in the pulmonary circuit compensated by hypertrophy of the right ventricle. The duration of childhood is rarely if ever sufficient for failure of this compensation, resulting in general venous stasis and transudation of serum. The form of the chest may become altered and approach more or less nearly the well-known barrel-shaped type: descent of the diaphragm and of the abdominal viscera lying beneath it is here more marked.

In interstitial emphysema we find small bubbles of air beneath the surface of the pleura, especially along the anterior borders of the upper lobes, and forming little chains in the course of the interlobular septa. These bubbles are movable and can be run together by pressure. Sometimes the pleura is raised up in larger blebs, the rupture of which may produce pneumothorax: this is, however, very rare in children. In connection with whooping-cough occasionally rupture takes place near the root of the lung, the air finding its way into the mediastinum and thence upward into the neck and face.

Symptoms and Course.—When we remember that in the great majority of cases the emphysema of children is not, strictly speaking, emphysema at all, but rather a hyperdistention of the lungs; that the causes to which it is due are usually in operation but a short time; that after these causes cease to act the distention generally soon passes away; and that these causes are apt to bring about other changes productive of symptoms, we can readily see that symptoms directly and clearly referable to the emphysema are very often either entirely absent or beyond our powers of recognition. Steffen has shown that even a true emphysema of short duration may present no symptoms whatever during life. These remarks apply to the whole period of childhood, but have special force with reference to its earlier years. The younger the child, the shorter time have causes in which to work and thus to give rise to symptoms and physical signs.

When symptoms are present they resemble those encountered in adults, and are, briefly, dyspnœa, constant, though varying in intensity in accordance with the extent of the accompanying bronchitis or other primary affection and the amount of secretion in the air-passages; cough, also varying in frequency and severity; and asthmatic paroxysms. To these may be added coldness of the extremities and increased dyspnœa on slight exertion.

Inspection shows pallor, with some cyanosis; a rounded chest with large antero-posterior diameter,—the frequency of combination with rachitic deformities must be borne in mind; labored respiration, expiration being especially difficult; and in younger children inspiratory retraction of the lower ribs, owing to the powerful contractions of the diaphragm acting on a yielding framework. Fürst has been led to consider as characteristic an expiratory distention above the clavicles during severe cough. In extreme cases the chest remains in the inspiratory position; it is lifted up, thus apparently shortening the neck; and the muscles attached to the clavicles may have an undue prominence. The motion of the chest as in one piece seen in sufferers advanced in life implies ossification of the costal cartilages, and is absent in children.

Percussion gives results far less distinctive than in adults, because of the small size of the chest and the great elasticity of its wall, which permits the transmission of vibrations from parts relatively distant from that over which percussion is practised. It goes without saying that percussion must be extremely gentle if the least value is to be attached to its results. And it may be stated, as a general rule, that the extent of the resonance is more characteristic than its intensity or quality. Thus what we look for is diminution in or loss of the cardiac dulness and a low position of the diaphragm.

The value of auscultation is very slight, certainly as affording indications of hyperdistention. The results which this method of examination does yield are to be connected with the primary or complicating affection. The vocal fremitus remains unchanged, a bit of evidence which may be of use in excluding pneumothorax.

Cyanosis, distention of the veins of the neck and trunk, epigastric pulsation, and accentuation of the pulmonic second sound in chronic cases indicate that there is heightened pressure in the lesser circuit which the right ventricle is scarcely able to cope with.

The course of the disease depends primarily on the nature of the underlying cause, on its severity and duration, and on the constitution of the child or its vigor at the time the illness began. From what has been said already it will be inferred that most cases run an acute course, some a chronic course but with ultimate recovery, while a few are permanent, with a tendency to gradual though perhaps very slow progression. In still others,—by no means rare, if we accept the views of Berkhart and Hertz,—recovery is not in reality so perfect as it seems to be, but the seeds of asthma and emphysema are implanted, to attain full growth many years later.

In the interstitial variety limited extravasations are usually, and extensive ones may be, completely absorbed.

Diagnosis.—Enough has been said to show that in a very large number of cases the diagnosis is to be reached only by inference from the presence of those causes which it is known are liable to be followed or complicated by the condition under consideration. In very acute cases a surprising degree of emphysema may be found after death without having been foreshadowed by symptoms during life. In chronic cases the recognition of the affection should involve no special difficulties to the observer, and is to be based on the same symptoms and physical signs as in older persons, due reference being had to those distinctions which have been already sufficiently detailed above. Pneumothorax is to be excluded by its limitation to one side of the chest, the respiratory excursion of which is lost, and by the lateral dislocation of the heart and the loss of vocal fremitus which it entails. The paroxysmal character of pure asthma and the entire comfort enjoyed in the intervals between the attacks rule out that affection. As regards the diagnosis of the primary disease, the reader is referred to the appropriate chapters of this work. Interstitial emphysema limited to the sublobular and subpleural tissue does not admit of clinical diagnosis. Theoretically mediastinal emphysema might be recognized, but practically it does not seem likely to be so often. Air in the subcutaneous cellular tissue of the face and neck, or even of the body, can be simulated only by serous œdema: the sudden advent and rapid spread of the former, with the peculiarities which it presents to touch, are distinctive.

Prognosis.—So far as acute emphysema can be said to have a prognosis, it is favorable. The emphysema itself never proves fatal, though it may co-operate with the underlying disease in bringing about a fatal result. Between the acute and chronic forms it is, of course, impossible to draw a sharp line, or to fix a duration beyond which ultimate recovery cannot take place. This must vary in different individuals and according to outward circumstances. While a genuine emphysema, considerable in degree, involves no real danger to life, certainly for a long period of time, it is at best an uncomfortable possession, carrying with it a liability to frequent catarrhal attacks, tending to grow worse, and seriously curtailing the activity of its owner. Emphysema, well marked and extensive, may under favorable circumstances and with good care pass off entirely in the end, even after it has lasted some time. The possibility of an apparent cure which years later turns out to have been delusive has been already spoken of, and is again alluded to because it is deemed important. The factors, therefore, which are to be taken into account in making the prognosis are the cause and its permanency, the previous health of the child, the extent of the changes as far as can be determined, the presence or absence of complications without as well as within the chest, and the willingness and ability of the parents to take such measures as are enjoined by an intelligent physician.

The belief formerly so generally entertained by the profession, that emphysema is a safeguard against tuberculosis, has grown much weaker of late years.

The prognosis of interstitial emphysema depends almost entirely on the cause which has given rise thereto. Reference is here made, of course, to emphysema appearing externally, or occupying the mediastinum.

Treatment.—Treatment should be in the first place prophylactic as far as may be. This means that delicate children, especially if there is any reason to think them hereditarily predisposed to emphysema, should be so managed as to avoid whooping-cough and attacks of bronchial catarrh. As regards the former, it is only possible to try to shun definite exposure; as regards the latter, quite as much care should be exercised in raising the standard of the general health, and thus lessening the liability to contract such attacks, as is devoted to more special precautions. When in spite of proper care children of this class fall ill with a respiratory affection, they must be more carefully treated and allowed to run less risk during convalescence than the robust.

That cardinal principle of treatment, to remove the cause, has a special applicability to a secondary affection, such as we have seen emphysema to be, at least in children. In acute cases the bronchitis is to be treated, the expulsion of excessive secretion is to be aided, violent cough is to be checked, and in general all those measures are to be taken which tend to shorten the course and mitigate the severity of the primary affection, whatever that may be. Emphatically here, the cause being removed, the effect vanishes.

In the more chronic cases we must still try to remove the cause, in the hope of preventing the condition from getting worse in case the changes are so pronounced that actual repair is not to be looked for. Chronic bronchitis, rachitic deformity of the chest, atelectasis, and the like must be combated. Hygiene in the broadest sense is of the utmost importance. Details are purposely avoided here, as they will be found in full where they more appropriately belong.

But the question arises, whether it is in our power to act directly on the hyperdistended or actually emphysematous lungs. The only means of which we have present knowledge consists in some form of pulmonary gymnastics, especially such as is promotive of expiration. This problem has been worked out much more carefully in Germany and on the Continent generally than in this country or in England. The work of Hauke, Waldenburg, and others is well known, and the pneumatic methods have proved of unquestionable service to adults. The difficulties of applying them to children, particularly very young ones, are obvious, but will doubtless be much lessened in time. There would seem to be no good reason why the pneumatic cabinet of Ketchum and Williams should not be applicable to some cases. The special act which seems most rational is expiration into rarefied air or its equivalent. But each case must be judged on its own merits, and mere routine in the use of pneumatic methods carefully avoided. A method which is admirably adapted to a pure emphysema of adults may be more harmful than helpful to a child with emphysema compensatory to or complicating rickets, atelectasis, or a lung bound down by pleuritic adhesions. As much active exercise in the open air as the climatic conditions and the strength of the child allow can do only good. Of course, the earlier after its origin the condition is subjected to proper treatment the better.

With reference to the avoidance of fresh catarrhal attacks, as well as with the object of keeping the child out in the fresh air as much as possible, it is sometimes advisable to insist on a change of climate. A climate which is either purely insular or quite removed from the sea-shore is, as a general rule, to be preferred. But great elevation, such as that of Colorado and New Mexico, is contra-indicated in emphysema.

Interstitial emphysema cannot be said to require treatment.

ASTHMA.

By FREDERICK C. SHATTUCK, M.D.

Definition.—Paroxysmal dyspnœa, sometimes periodic, with entirely or comparatively free respiration during the intervals between the attacks.

History.—Before the days of Laennec, the term asthma covered embarrassment of the respiration, with wheezing, almost irrespective of the cause. When physical examination of the chest during life was carefully checked by dissection after death, it was found that in most of the cases presenting asthmatic symptoms, more or less well marked but varying anatomical lesions were detected. A natural reaction followed, and leading authorities held the non-existence of asthma as a distinct disease. Further observation, however, aided by Reisseisen's discovery of the presence of muscular fibre even in the smallest bronchi, and the proof of their electric contractility by Longet and Williams, led to the abandonment of these views, and to the recognition of spasmodic asthma as an independent affection. Such it is to-day generally, though not universally, held to be.

Etiology.—It is customary among medical writers to distinguish between primary, pure, uncomplicated, spasmodic, or bronchial, and secondary or complicated asthma. Whatever may be the case with adults, it seems to the writer that this distinction is less applicable and hence less important with children: the reasons for this opinion will appear later. Here a division is made simply into predisposing and exciting causes.

1. *Predisposing Causes.*—First of these is hereditary influence, traceable in too large a proportion of cases to allow us to suppose it to be mere coincidence. In rather more than two-fifths of two hundred and seventeen cases, Salter finds distinct traces of inheritance, direct or lateral, immediate or remote. It is probable that asthma is to be regarded as one of the many and various manifestations of what is called to-day the neurotic temperament or constitution,—a tendency to disordered nervous function under the operation of secondary causes which in most individuals are totally inadequate to produce such results. The exciting causes are wide-spread and frequent, whereas asthma is, comparatively speaking, rare.

The heredity of the affection is furthermore shown by the age at which it first appears. Salter finds that more cases originate during the first decade than during any other equal period of life, that in these hereditary influence is usually present, and that fewer cases originate between ten and twenty than in any other decennium. He has seen asthma in infants of fourteen and twenty-eight days, and ten cases under one year of age. The earliest ages at which Politzer has seen the affection are ten and fifteen months. Soltmann believes that some of Salter's cases were really thymic, not bronchial, asthma.

Males are much more liable than females,—a fact which does not seem to agree well with the theory of the nervous origin of the malady. Salter and others suggest, as an explanation, that males are far more exposed to the various exciting causes,—the weather and its vicissitudes, for instance. While this explanation is very likely correct as regards adults, it does not seem to be so as regards children. Up to the age of ten there is not very much difference in the degree of exposure to which the two sexes are subjected, and we find that sixty-three of Salter's cases originated before that year. Of these forty-six were boys, seventeen girls. It seems odd that this analysis, which the writer has worked out from the table, has not been made before. The reason lies perhaps in the fact, which is a striking one, that, even in the leading text-books on children's diseases, asthma has received surprisingly little attention.

It is more common in the upper than in the lower classes, probably because the nervous system is more sensitive in the former; and it is said by Soltmann to be, like diabetes, particularly common among the Jews.

2. *Exciting Causes.*—Those causes which, acting on a subject predisposed, excite asthmatic paroxysms may be divided into (1) those which act directly on the lungs and (2) those which act primarily on a distant organ or part.

(1) Chief among these is bronchitis, either simple or as a manifestation of whooping-cough or measles. A sharp attack of bronchitis may in some children, especially if it involves the smaller tubes, give rise to asthma or dyspnœa greatly resembling that characterized as asthma. At each subsequent attack of bronchitis, however slight, the asthma may return. Or, what is more common, the long-standing cough and bronchial irritation of pertussis or measles may produce, particularly in scrofulous subjects, enlargement of the bronchial glands, the pressure of which on the pneumogastrics is in some children sufficient to excite paroxysmal dyspnœa. Stress should be laid on the word *some*, as it is probable that only a small proportion of those whose bronchial glands are enlarged ever manifest this symptom. There must be a predisposing as well as an exciting cause. Another mode in which chronic bronchitis may prepare the way for asthma is by the production of emphysema, more or less well marked clinically and greater or less in degree. Again, attacks are traceable to atelectasis originating in rachitic deformity of the chest or lobular pneumonia. These are, of course, among the causes usually classed as secondary.

The pressure of mediastinal tumors of non-glandular origin,—aneurism in adults,—of goitre, rare in this country, and of enlarged cervical glands, may also be causative.

Next come irritants, very various in kind, and of varying obnoxiousness to different persons. That which is sure to bring on an attack in one may have no appreciable influence over another. Among these are dust and pollen (see Hay-Fever), fog and smoke, fumes and vapors, emanations from animals, and climatic influences. Many curious facts could be cited under this head, such as the immunity of one person in the smoky city, of another only in the country,—perhaps, as with hay-fever, not in the country generally, but only in certain localities which may be very circumscribed; the inevitable paroxysm brought on in some by exposure to powdered ipecac, by the near presence of a cat, dog, or horse, or by feathers,—perhaps only by those of a certain kind. We are utterly unable to explain these facts, and hence speak of idiosyncrasy. They are encountered oftener in adults than in children, though it must not be thought that the latter are quite exempt from these peculiarities.

(2) The distant irritations which, generally acting through reflex paths, are recognized as provocative of attacks may be divided into—

(*a*) Those acting on the nasal passages. Voltolini first called attention to the relations of polypi of the nose and asthmatic attacks, and of late years the causative influence of other affections of the nasal mucous membrane has been demonstrated often enough in adults by Hack, Mackenzie, Roe, and others. We have not found cases of this class in childhood reported in literature.[1]

(*b*) The starting-point may be the stomach or the intestine,—peptic asthma. Many asthmatics learn that they must be careful in their diet generally, must avoid certain articles of food, or must eat sparingly and simply at certain periods of the twenty-four hours,—usually towards night. Intestinal worms are also set down as a cause.

(*c*) More common than either of the above-named causes, certainly during childhood, are some skin-affections, notably eczema and urticaria,—herpetic asthma. West, as quoted by Eustace Smith, says he has "never known eczema to be very extensive and very long continued without a marked liability to asthma being associated with it." The two affections may alternate, or they may be coexistent, and the cure of one may be followed by the disappearance of the other. We shall return later to the relations of urticaria and asthma. Salter tells of a man who could produce an attack at will by applying cold to the instep.

The above are the chief distant reflex causes. The irritation may, however, act directly on the central nervous system, as through a poison circulating in the blood. Uræmic, gouty, and saturnine asthmas belong in this class. So also probably does cardiac asthma, the exciting cause in this case being perhaps carbonic acid. Trousseau tells of a boy of five whom he saw in well-characterized fits of asthma. Two years later the boy had typical gouty arthritis, and during its continuance was free from his asthma. The writer has been no more successful than Soltmann in finding a recorded case of asthma in a child due to purely emotional causes.

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[1] That hypertrophy of the tonsils may provoke attacks is shown by their cessation after the redundant tissue is removed.

There remain, finally, a certain number of cases in which no definite exciting cause can be made out.

Pathology.—The clinical facts and the lack of unanimity in their interpretation among careful and experienced observers go far to convince the writer that asthma is, certainly in the great majority of cases, a symptom rather than a disease. This is the view ably advocated by Berkart, and the more thoroughly one studies the literature of the subject the more one is drawn into agreement with him. Moreover, the asthma of childhood seems to us to lend particularly strong support to this view. In the first place, the very term asthma is employed loosely, even by modern writers, some giving it a much wider acceptation than others. Trousseau, himself a sufferer from asthma, relates the case of a child, stating that he afterwards saw others similar, with alarming dyspnœa which he attributed at first to catarrhal pneumonia. It was not till after he had watched the second attack, and twice seen the child recover from a condition which in his experience was rarely if ever recovered from, that the idea of the asthmatic nature of the seizure entered his mind. In other words, the peculiarity lay in the recovery, not in the features of the dyspnœa. There would be no difficulty in adducing abundant evidence bearing on this point, were this the place for it. Another argument, and a strong one, which leads us to think that asthma is a symptom, is the fact that in children it is so often entirely recovered from at or about the period of puberty, if not still earlier. It is, that is to say, largely dependent on some removable cause. We see recovery also in adults, but this result is less frequent and less complete. Salter and Williams, both believers in the not uncommon occurrence of asthma as an independent disease, state that in eighty per cent. of the cases developing in childhood bronchitis appears to be the starting-point. The whole etiology of the affection goes to show that the first factor to take into account is predisposition, varying in degree in different individuals. The second factor is some deviation from perfect integrity of structure in some portion of the air-passages, oftentimes one which we are not at present able to detect; or an undue excitability of the mucous membrane which leads it to resent irritation, manifold or single in kind; or, finally, a distant irritant which, owing to individual peculiarity, is reflected to and again from the respiratory centre. The greater the predisposition, the less does the exciting cause need to be. Perhaps in those cases, the existence of which we are far from wishing to ignore, where the most careful study during life, with or without minute examination after death, fails to reveal any exciting cause, or only such changes as are more probably secondary than primary,—perhaps in these there may be molecular or vascular changes from time to time in the respiratory centre. Is it an unjustifiable hypothesis that asthma may be to the respiratory centre or centres what epilepsy is to the motor regions of the cerebral cortex?[1] Holding, then, that asthma is at all ages, but especially in children, a symptom rather than a disease, the next question to consider is, wherein lies the pathology of this symptom?

There is more agreement as to its being a neurosis of the vagus than there is as to the mechanism through which the neurosis is manifested. The very divergence of opinion here is an indication that no one theory satisfactorily explains all the facts, and inclines one to think that the pathology is not uniform in all cases.

There are three leading views, each of which has its adherents, though some of these do not claim a monopoly of truth for their ideas.

(*a*) The bronchial-spasm theory is the favorite at the present day, and counts among its supporters Laennec, C. J. B. and C. T. Williams, Trousseau, Salter, Biermer, and Thorowgood. This theory explains the suddenness with which the dyspnœa may come and go, the rapid changes in the seat and number of the adventitious sounds, and the effects of certain drugs, especially narcotics and antispasmodics.[2]

There is a difficulty about this explanation to those who, with Biermer, hold that the dyspnœa of asthma, as of all other conditions in which the obstruction is seated in the finer tubes, is always expiratory. If the bronchial-spasm theory is true of all cases, it would seem that both respiratory acts ought to be equally difficult. Biermer replies to this objection, "When the bronchi are spasmodically contracted, they are subjected during expiration to the general pressure of that movement plus the pressure of the spastic contraction of the bronchial muscles. The walls of the bronchioles being soft and compressible, the expiratory pressure, instead of overcoming the obstruction and opening them, would tend to close them all the more tightly."

(*b*) Wintrich originally propounded the theory of spasm of the diaphragm, a view shared by Bamberger and Riegel. To this Eichhorst answers that the action of the diaphragm may be observed during many a paroxysm, and that prolonged tonic spasm of this muscle is not compatible with life.

(*c*) Weber, Störck, Sée, Sir Andrew Clark, and others reject more or less completely the bronchial-spasm theory, and attribute the symptoms to swelling of the mucous membrane of vaso-motor origin. Clark considers this analogous to urticaria, and lays stress on the cases in which asthma and urticaria are associated or replace each other. Störck was led to adopt this theory by laryngoscopic examinations made during paroxysms. He found the trachea congested, and inferred that a similar condition was present throughout the bronchial tract. Glasgow, of St. Louis, has also used the laryngoscope, but finds a pale mucous surface, while he attributes the swelling and dyspnœa to vaso-motor spasm, not relaxation, and saturation of the tissues with liquor sanguinis. He supports his position by the prompt and remarkable effects of the nitrites, the relaxing influence of which over the walls of the smaller arteries is well known. It is interesting to note that Fraser, who has studied the action of these drugs carefully in asthma and bronchitis, is an adherent of the bronchial-spasm theory.

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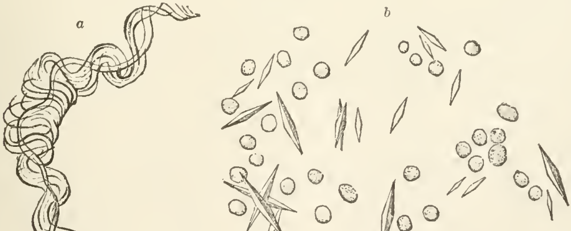
[1] Salter relates the case of a man in whom asthmatic replaced epileptic attacks, and, like the latter, were preceded by an aura. The points of resemblance between asthma and epilepsy did not escape this acute observer.

[2] Morton (British Medical Journal, 1887, ii. 159) calls attention to the similarity between false croup and asthma, and quotes West's observation that children who have had croup in early years are prone later to suffer from asthma. Morton queries whether hay-fever, asthma, and croup are not merely different expressions of the same state.

The fluxionary-hyperæmia theory is largely based on the existence of cases in which every attack of bronchitis markedly increases the liability to asthmatic paroxysms and in which more or less emphysema is present,—the wet asthma of the older writers. The asthma of the later stages of hay-fever also belongs here. In this complaint we have hyperæmia of and flux from the visible mucous membranes, and, there is good reason to think, also from the bronchial tubes. In congestive asthma the dyspnœa diminishes as secretion becomes free. There is some doubt as to whether cardiac asthma is dependent on carbonic-acid and uræmic poisoning, or on passive congestion of the bronchial mucous membrane.

Leyden thinks that the sharp-pointed crystals which he has described as present in asthmatic sputum irritate the nerve-filaments and thus excite spasm. These crystals are found, however, in various pulmonary affections unassociated with asthma. The same is true of the spirals with which Curschmann's name is linked. They indicate the presence of what he calls "bronchiolitis exsudativa," and are to be seen in pneumonia.

FIG. 1.



Curschmann's spirals (*a*) and Leyden's crystals (*b*).—After Strümpell.

To close this brief and incomplete exposition of the pathology of asthma, the words of Berkart are appended, the italics being his: "*Asthma, therefore, is only one link in a chain of quasi-independent affections, which commences with inflammatory changes of the pulmonary tissue and terminates with emphysema or bronchiectasis.*"

What has been said above indicates clearly enough that asthma itself has no pathological anatomy, and also what the changes are on which the paroxysmal dyspnœa depends when these changes are sufficiently developed for us to detect them. In children, at least, we have to do with bronchial or pulmonary inflammation, emphysema, or enlargement of the bronchial glands, in the great majority of cases.

Clinical History.—The symptoms and physical signs vary, in some important respects, according as the particular case comes under the one or the other of two main classes, though in both alike the attack usually appears during the evening or night, often waking the child from sleep. In both, also, the child sits up in bed, is restless, and instinctively seeks to overcome the struggle for breath by grasping the bedclothes or some other object, thus facilitating the action of the accessory muscles of respiration.

In the first and more typical class the child was during the previous day and evening apparently as well as usual, though it may have had symptoms of a trifling cold. The attack sometimes attains its full severity very rapidly, sometimes more gradually. The face is pale, cyanotic, and anxious; the skin moist and cool; there is no fever; the pulse is rapid and often irregular. The respiration is slow and labored, expiration being much prolonged; the chest is fixed in the position of full inspiration, with a low diaphragm; percussion-resonance is increased in intensity and area; on auscultation the respiratory murmur is much enfeebled or absent, and sibilant and sonorous râles are heard everywhere. Cough, if present, is short and dry. Towards the end of the attack, if the child is old enough to expectorate, a little tough and viscid white mucus may be expelled. The fit, after lasting a variable time, may go nearly or quite as rapidly as it came, the child falling asleep, and waking in the morning about as well as usual. A recurrence may take place the following night or nights, or may be delayed a variable length of time.

In the second class—the more distinctly catarrhal—the child has suffered perhaps for several days from bronchitis of more or less intensity; has fever; and the dyspnœa presents less of the expiratory type, perhaps is rather inspiratory. The chest is not hyper-resonant; the diaphragm is not depressed; the soft parts above and below the thorax are drawn inward on inspiration, the rate of which is quickened, perhaps to fifty or more. In addition to the coarse dry râles, finer and moist râles are heard, especially over the bases. The cough is less dry and more frequent. The attack passes away gradually, with lulls and exacerbations during several days or more. A fresh attack of bronchitis brings the asthma anew.

The difference between the two varieties depends on the bronchial inflammation, and the blocking of the tubes by secretion which characterizes the latter. Trousseau called attention to the frequency of these cases in children. The first case which he saw has been already alluded to in discussing the pathology. Their existence seems to have been overlooked by Biermer, who maintains that the dyspnœa of asthma is always purely expiratory. Politzer and others describe cases in which a remarkable periodicity characterized the recurrence of the paroxysms.

Diagnosis.—This may be difficult, especially in cases of the second class and in the first attack. A previous history of similar fits is, of course, of great value for diagnostic purposes. Careful attention to the history and the presence of such symptoms and physical signs as have been sketched above, with, in doubtful cases, the subsequent course of events, will generally reveal the nature of the affection.

Eustace Smith has given us valuable instructions for the detection of swollen bronchial glands. "Pressure on the descending vena cava or the left innominate vein gives rise to a hum, and on the pulmonary artery to a systolic murmur heard best at the second left interspace. But long before the ordinary signs of pressure on the vessels can be detected, we can induce pressure on the vein if the bronchial glands are enlarged. This sign is one of the earliest indications of disease of these glands. Thus, if the child be directed to bend his head backward upon his shoulders so that his face is turned upward to the ceiling, a venous hum, which varies in intensity according to the size and position of the swollen glands, may be heard with the stethoscope placed upon the upper bone of the sternum. As the chin is slowly depressed again, the hum becomes less distinctly audible, and ceases shortly before the head reaches its ordinary position. The explanation of this phenomenon appears to be that the retraction of the head tilts forward the lower end of the trachea. This carries with it the glands lying in its bifurcation, and the left innominate vein is compressed where it passes behind the first bone of the sternum. I believe this explanation to be the correct one, for in cases of merely flat chest, where there is no reason to suspect enlargement of the glands, the experiment fails. Nor, again, can the hum be produced in a healthy child by the thymus gland. This gland lies in front of the vein immediately behind the sternum. Enlarged bronchial glands lie behind the vessels in the bifurcation of the trachea. A swelling in front of the vessels does not appear to be able to set up pressure upon the vein when the head is bent backward in the position described."

Other signs of enlargement are dulness over the first bone of the sternum or between the scapulæ, only to be made out when the glands approach the inner surface of the chest; and dilatation of the superficial veins of the thorax, or slight œdema of the face, perhaps unilateral, from pressure on the venous trunks within the chest.

The absence of stridor, of ringing cough, of patches of membrane in the fauces, and the predominantly expiratory character of the respiratory difficulty, exclude croup, true and false. In cases of obstruction of a main bronchus by a foreign body, the disparity in intensity of the respiratory murmur on the two sides is diagnostic.

Of course other causes of paroxysmal dyspnœa, such as retropharyngeal abscess and cardiac or renal disease, must be thought of and excluded.

The former can be felt by the finger in the throat, the latter are to be detected by the usual methods of examination.

Prognosis.—This is, as has been stated in another place, as a general thing good,—better than in adults,—and for the reason that the attacks depend so often on bronchial-gland enlargement or some other cause which can be influenced by treatment or which may disappear of itself.[1] Much depends on the constitution of the child; more, perhaps, on the care and advantages which easy circumstances of the parents allow it to enjoy. A strong hereditary predisposition does not in itself preclude recovery. If notable emphysema has already developed, and especially if it has existed some time, the outlook is less favorable. In general, the prognosis may be said to depend on that of the underlying cause: if this is removable, it is good; if not, it is bad. This fact affords an additional reason, if such be needed, for the establishment of an accurate and thorough diagnosis,—a remark which applies with at least equal force to

Treatment.—This is to be discussed under three main heads: (1) prophylaxis, or the prevention of the affection in those presumably predisposed; (2) cure, or the prevention of the recurrence of attacks in those who have already experienced them; and (3) palliation, or the treatment of the paroxysm itself.

(1) *Prophylaxis.*—Where there is a well-marked tendency to asthma, emphysema, glandular enlargements, or perhaps to neuroses, special care should be exercised in the management of the children. The wisdom and watchfulness of the mother, aided by a sensible medical adviser, will be shown as much in the avoidance of coddling as in the taking of all proper and reasonable precautions against undue exposures and in the maintenance of sound hygiene. Out-door country life is desirable for such children when it can be secured; and, with this, woollen clothing, a simple and highly-nutritious diet, and careful attention to house ventilation, both of living- and of sleeping-rooms, should be combined. Exposure to whooping-cough and measles and other infections is more easily guarded against in the country than in the city. Should the child acquire these diseases, they ought to be so treated as to shorten their course and diminish their intensity as far as may be, in order that structural changes in the lungs, however slight, and swelling of the bronchial glands, may be avoided.

(2) *Cure.*—Accurate diagnosis is the first prerequisite. But, in general, good hygiene is of the utmost importance, and of more value than all other therapeutic measures put together. The latter can be made, however, to render much service if skilfully and persistently applied. Enlargement of

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[1] The popular idea that asthma in children is curable is well illustrated by the domestic treatment practised in some of the country districts, for a description of which the writer is indebted to Dr. Morrill Wyman, of Cambridge. The child is placed standing against a tree (a maple is preferred); a hole is bored just above the head; the nails and a lock of hair are cut and placed in the hole, which is then plugged. As the child grows above the plug, the asthma is left behind.

the bronchial glands calls for cod-liver oil and the iodide of iron if the tongue is clean and the digestion fairly good. But it is not infrequently necessary to precede the administration of these remedies by a bitter tonic, with perhaps a mineral acid, and by mild laxatives. The appetite, digestion, and nutrition in general are thus stimulated, and the absorption of glandular hyperplasia and of the remains of an inflammatory process in the lungs is promoted. Potassic iodide, continued steadily for weeks in as large a dose as is well borne, yields sometimes brilliant results in the asthma of all ages. Arsenic, again, is of great value in some cases, and is generally well tolerated by children.

Yandell[1] reports the following case, the result in which led him to employ the same treatment in eight others with a good measure of success. A girl of six had suffered for two years from asthmatic attacks occasioned by "colds," the provocations to which were becoming smaller. Fresh air and sponge-baths were ordered; ten to twelve grains of potassic bromide were given on rising and on going to bed, and also at the latter time atropia, grain $\frac{1}{125}$; and a cough-mixture containing a little opium was prescribed whenever the child showed symptoms of a cold. This treatment was kept up for three months, with a diminution in the frequency of the attacks. The remedies were then omitted for two weeks, and resumed again uninterruptedly for four months. About this time, after wetting her feet, the child had another (but her last) attack. During the next four months the medicines were given fifteen days in each month: they were then omitted for several months, and, finally, were administered steadily for sixty days. Sharp catarrhal attacks have come from time to time, but the asthma has never returned. Of the eight other cases treated in the same way, three were ten and two were eleven years old, and the ages of twelve, thirteen, and fourteen were each represented by a case. All but two recovered, and in these the treatment is said not to have been thoroughly carried out. None were dismissed under fifteen months of treatment, and two were treated two years. In five the affection was hereditary, and eight of the nine were neurotic children. These cases are detailed here because the results were good and there are few observers who report so many cases. It is true that all but one of the children were approaching the age when the symptom may disappear without special treatment. It should be added that in no case was there change of climate. But hygiene was, apparently, not neglected.

The last meal of the day should be particularly light and simple.

Almost all asthmatic children are better off in the country or at the seashore, due reference being had to individual peculiarity in making a selection between the two. One can expect more benefit from sea air and bathing in cases attributable to enlargement of the bronchial glands. The asthma of adults is cured in a large number of instances by residence at high altitudes, as in Colorado, for example.[1] Dr. Fisk, of Denver, informs the writer that he is neither himself cognizant of a case of child asthma in that city, nor has he been able to learn of a case on inquiry among his professional friends. The prognosis of child asthma is so good that great sacrifices for the sake of climatic change are very rarely called for. Removal to a short distance and for a limited period will serve every purpose in most cases. Much emphysema is a contra-indication to high altitudes. The pneumatic treatment has less applicability to children than to adults.

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[1] American Practitioner and News, 1886, vol. i. p. 132.

(3) *Palliation.*—The treatment of the paroxysm varies according as the special case is of the purely spasmodic or of the catarrhal type, and in the latter according to the amount of secretion. In typical spasmodic cases the greatest variety of remedies is advised, no one proving equally satisfactory in all. Nearly all these remedies are to be classed among either the narcotics or the antispasmodics. It is, of course, only in an extreme case and in the later years of childhood that morphine hypodermically should be used. Chloroform and ether arrest the fits, but only temporarily, the attack returning as the effects of the anæsthetic pass off. Chloral with or without potassic bromide renders excellent service, in doses proportioned to the age of the child. Nitre-paper is a time-honored remedy. Inhalation of the iodide of ethyl is praised by Sée: ten minims can be safely used for a child. Belladonna and lobelia are highly spoken of by some, but are uncertain in their action. The former owes its popularity to the great authority of Trousseau. The patented powders and pastilles which are in such vogue among the laity, and which it must be confessed are often efficacious, contain nitre, stramonium, and lobelia. Pilocarpine is said by Berkart to act well in children, an eighth to a tenth of a grain being given under the skin to a child of five years. The nitrites, if used at all, should be administered with caution, on account of our limited experience with them at this time of life.

In catarrhal cases where the symptoms and physical signs indicate the presence of abundant secretion, a simple emetic, such as ipecac, will clear out the bronchial tubes, relax spasm, and materially relieve the breathing. No true asthmatic paroxysm can withstand the depressant effect of nausea.

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[1] Dr. Solly, of Colorado Springs, writes that neither he nor the other leading physicians of that place have ever known asthma develop there in a child. Dr. Solly himself recalls three children coming there with asthma. Two of these had no apparent complications and went away seemingly well. He has not heard of them since. The third was a girl of eight who came from England with bronchitis and asthma, remained two years, and has since then remained well in England.

HAY-FEVER.

By FREDERICK C. SHATTUCK, M.D.

Synonymes.—Hay- or Rose-cold, Summer or Autumnal catarrh, Hay-asthma, Periodic coryza, Coryza vasomotoria periodica, Rhinitis sympathetica, etc.

Definition.—An affection, as a rule, annually recurring and periodic, characterized by irritation and redness of and flux from the mucous membranes of the eyes, nose, throat, and bronchi.

The term "Hay-Fever" is taken as the heading of this article for the reason that it is in such general use, although its unsuitability is recognized by all. The name is simply an illustration, of which there are so many in medical nomenclature, of the way that a term denotive of a mistaken etiological or pathological theory gains so firm a foothold that it is preserved long after its falsity has been proved. Beard was not able to trace the exact origin of the term, but thinks it must be credited to the laity, who noticed the coincidence in time between the onset of the symptoms and the haymaking season. Wyman's name, "autumnal catarrh," is indicative of his idea that the August are different from the June cases, and that there is no middle form. The investigations of Beard and the observations of later writers would seem to necessitate a modification of these views.

History.—To Dr. John Bostock belongs the credit of having first really described this interesting disease; and, etymologically, *disease* it may be called, there being many of far greater gravity which entail much less discomfort to their owners. Dr. John N. Mackenzie, of Baltimore, who has made most important contributions to our knowledge of the ailment under consideration, has with great industry searched ancient medical literature and found here and there clear evidence that the affection was existent and recognized, though very imperfectly, in previous centuries. Constant de Rebecque, writing in 1691, attributed his symptoms after thirteen years' experience to "something which flows from roses, which stings the nose and by means of tiny prickles produces a solution of continuity imperceptible to the naked eye." He may thus be regarded as the father of the pollen theory. Bostock was himself a sufferer, described his own case in 1819, and wrote again in 1828, but more at length, proposing the name "catarrhus æstivus." He was followed by several other English writers, and in 1862 appeared the treatise of Prof. Phoebus, of Giessen, founded on replies to a circular comprising seven questions which he sent out to physicians. In 1872, Dr. Morrill Wyman, of Cambridge, in common with many members of his family a sufferer, published his highly interesting and important monograph based on an analysis of eighty-one cases. In 1873 Blackley's work appeared in England, and in 1876 the press brought forth an exhaustive treatise from the prolific pen of the late Dr. George M. Beard, whose second circular, containing fifty-five questions, elicited replies from two hundred of the afflicted or their professional attendants. Since then no systematic treatise has been published, though important contributions to our knowledge of the pathology and treatment have appeared in the medical journals, embodying the experience of Daly, Roe, John N. Mackenzie, Bosworth, and others.

Etiology.—As in asthma, so in hay-fever, though there would seem to be less room for cavil with reference to the latter, the chief predisposing cause is the neurotic temperament, which, amid the excitements, hurry, and strain of our complicated modern life, seems to be constantly forming new centres of development. Environment may increase a tendency in an individual, or may, perhaps, produce it anew; when produced it is frequently transmitted to descendants. The cases in which several members of a family are hay-fever subjects are far too common to be attributed to mere coincidence. Vast numbers of persons are exposed to the exciting causes of this peculiar affection without ever suffering from it. Another predisposing cause seems to lie in a more or less unhealthy condition of the mucous membrane of the upper air-passages, especially the nose. Males are rather more liable to suffer than females. The affection is much more common in this country and in England than on the continent of Europe, and here the Anglo-Saxon race seems to suffer more than the foreign population. Mackenzie, of Baltimore, has seen three cases in negroes, and there can be no doubt that both Wyman and Beard were mistaken in thinking the affection to have such narrow race- and class-limitations as they did. Now that the attention of the profession has been awakened to the subject, cases are not infrequently encountered in dispensary and hospital outpatient room practice. At the same time, the causes which produce the neurotic temperament operate more forcibly on the wealthier and more highly educated classes, and have been longer in operation on the native than on the foreign population.

The frequency of the affection in children is shown by the following figures, which indicate the age when the symptoms first appeared. Wyman, 72 cases: under 10, 11 cases, 15 per cent.; between 10 and 20, 15 cases, 20 per cent. Beard, 192 cases: under 10, 34 cases, 17 per cent.; between 10 and 20, 29 cases, 19.98 per cent.

The chief exciting causes are season, irritants, sunlight, and heat.

Although it is true that in some individuals attacks may come on at any season of the year, the fact remains that the period of danger for the vast majority is comprised between the months of May and September, both inclusive. We can, indeed, go farther, and broadly distinguish an early and a late form. The former, the more common in England, usually begins in May or June, and has been called "rose-cold"; the latter is apt to appear towards the end of August, the "autumnal catarrh" of Wyman. Beard seems to have been the first to demonstrate the existence of a form intermediate in point of time. This causative influence of season is undoubtedly closely bound up with the other excitants mentioned above, especially heat and the presence of mechanical irritants in the atmosphere, such as inorganic dust of all kinds and the pollen of plants and trees. But it should be remembered that in exceptional cases the attacks may persist into or even recur in the winter.

The irritating excitants are many in number and various in kind. Beard gives a list of some thirty, without pretending that it is complete. Of course it is not to be inferred from this that each and all of these irritants are equally obnoxious to all persons. There is here a considerable range of individual peculiarity. Still, it may be stated as a broad fact that alleviation of symptoms and freedom of the atmosphere from dust are most intimately connected. Indigestion and over-exertion distinctly tend, in the opinion of some, to induce or aggravate the attacks. The influence of warmth and sunlight is well recognized by many, who, if they cannot flee from their torment, seek to mitigate it as far as they can by seclusion in closed and darkened rooms during the heat of the day and while the sun is high. This is, of course, impracticable for most sufferers, who can nearly as well seek a region of immunity.

Locality is a factor which one hardly knows whether to class as predisposing or exciting; it is also a factor which diminishes in importance as the affection receives more careful and more wide-spread study. Beard shows that Wyman is inclined to confine the disease within too narrow geographical limits, and later writers show that it is more wide-spread than Beard seems to have imagined. There are, however, in the Northern and Eastern States certain sharply-limited areas which are well known as conferring immunity. The attack is prevented by moving to these before the symptoms appear, or is promptly cut short if they have already begun. Moreover, the symptoms appear or recur if the refuge is quitted before the period has elapsed during which they last when no climatic change is made. These regions of exemption are chiefly mountainous districts, and limited portions, only, of them. The favorite resorts are certain localities in the White and Catskill Mountains; but a place which grants perfect immunity to one person does not necessarily grant the same to another, and a short drive of a few miles may make all the difference between comfort and misery. There is no fixed elevation at or beyond which relief is sure, —though the influence of elevation cannot be denied. This remarkable immunity is doubtless due in great measure to coolness and relative freedom from dust and vegetation. A gentleman tells the writer that even at the

Isles of Shoals a wind from the mainland, seven miles distant, brings on his sufferings anew. The open sea is the only resort which can be depended on for relief as universal as it is complete.

Pathology.—Hay-fever is primarily a neurosis,—indeed, a pure neurosis in all cases in which there are no notable nasal lesions persistent between the paroxysms. The undue excitability may have its seat in the terminal nerve-filaments of the nasal passages, in the centres directly or indirectly connected with these filaments, or in both at once. The most prominent symptoms point to great vaso-motor disturbance: how much of this is purely reflex, how much is not, we have no means of accurately determining. That hay-fever is a neurosis is proved by its utter want of constant pathological lesions; its hereditary character; the constitution of those affected by it; its dependence on exciting causes to which vastly greater numbers of persons are exposed than are affected thereby; its analogy with other affections largely of a neurotic nature, such as asthma, false croup, and sick headache; its annual periodicity, the subjects of it being perfectly well and manifesting no unusual susceptibility to ordinary colds in the intervals between the attacks; the close similarity in its symptoms in different individuals, while there is a decided variation in the special exciting cause; and the rapid and complete subsidence of the symptoms on change of locality, perhaps trifling in degree.

Symptoms and Course.—The date on which the symptoms begin to recur each year is in some cases absolutely definite, though in the large majority there is a variation of a few days or more. In some there is a prodromal stage lasting one or two weeks, during which there may be more or less nervous irritability, or alternating sensations of heat and cold, or a feeling of lassitude. In other cases a prodromal stage is wanting. Although the symptoms of the disease are similar in all sufferers, they are far from being identical: there are considerable variations not only in the general intensity of the process, but also in its special localization. The intensity of the attacks also varies in the same person often from year to year.

The first symptom, and one which at once distinguishes hay-fever from a common cold, is usually itching about the roof of the mouth and an uneasy sensation in the Eustachian tube; this is soon followed by frequent sneezing; paroxysmal obstruction of the nostrils of short duration, at first confined to the early morning, but afterwards recurring later in the day; a watery discharge from the nose, especially on lowering the head; attacks, also paroxysmal, of irritation of the eyes, with itching of the lids, especially at the inner canthus, inducing the patient to rub them vigorously; redness and swelling of the face in the morning; and impairment or even loss of the special senses of smell, taste, and hearing. Itching of the scalp and of the skin of the back or chest, a tendency of the skin to become easily excoriated and, when excoriated, to heal slowly, and more or less general depression of the system, with lack of appetite and quickening of the pulse-rate, are often experienced during this period, which lasts ten days to two weeks. The irritation now extends to the bronchial mucous membrane, exciting a short and annoying cough, which results in but little expectoration, and that of transparent glairy mucus. The cough is worse in dry than in damp or wet weather, at night than during the day, and increases for a week or ten days. During the fourth week the early symptoms are apt to diminish, but the cough persists, and asthma, if it comes at all, now appears on the scene, intensifying the misery of the night. During the fifth and sixth weeks there is a gradual decline, and the patient soon after regains his wonted comfort and strength until his time of trial comes round again the following year. The above is, very briefly, the course of the disease.

Diagnosis.—It is only in the first attack that this can present any real difficulty to the observant practitioner. The season of the year, the family and previous history of the child, the character and sequence of the symptoms, their variation from day to day and mitigation by damp weather, the failure of well-directed treatment to bring more than palliation, and the six weeks' course of the affection, are, all taken together, distinctive enough. Finally, the rapid, not gradual, cessation of all symptoms after removal to a recognized hay-fever resort, whatever the period of the disease, and their prompt recurrence on leaving the same, provided that the time-limitation has not been reached, may be added.

Prognosis.—As regards expectation of life, this is good. Hay-fever patients seem to live as long as those who are free from the infirmity. As regards a cure of the morbid susceptibility, the outlook is different. In a recent note to the writer, Dr. Wyman says, "So far as I have observed and read, I think the *entire* disappearance of the annual attacks of hay-fever in those with whom it began in childhood is very rare; I have never seen it. The attacks, however, may diminish, and sometimes exhibit an early and a late form, with, I think, a diminished severity." John N. Mackenzie, on the other hand, tells me that he considers the prognosis in children good for a considerable number of cases. The cures which are reported, whether of children or of adults, depend chiefly on local treatment of the nose. It is not surprising that hay-fever should differ from ordinary asthma in the young, as regards frequency of disappearance, either spontaneously or under purely general treatment. In the latter the prime cause may be said to lie, certainly in as large a number of cases as recover, in anatomical changes in the bronchial glands or some portion of the respiratory tract; the subordinate cause, in a peculiarity of the nervous system of the individual. In hay-asthma, on the other hand, we frequently have no reason to think that there are any actual lesions except those which appear with the onset of the symptoms, to vanish entirely with the subsidence of the latter.

Treatment.—If there be a method of treatment which is prophylactic in the sense of preventing the development of hay-fever in one presumably predisposed, we are entirely ignorant of its nature. Although the susceptibility is, we have good reason to think, often inherited, it is not necessarily so, and it would be a difficult matter to feel sure that measures adopted in any given case before the appearance of symptoms were the cause of their non-appearance.

It is in our power, however, to prevent the recurrence of the disease after it has developed, by annual change of residence to a locality, whether an island, the sea-shore, or a mountain-resort, which experience shows gives immunity to the particular individual. It is desirable to go shortly before the time of the expected attack and to remain at least six weeks, after which time the danger for that year is practically over. The leading White Mountain resorts are Bethlehem, Jefferson, Gorham, the Twin Mountain House, and the Glen. The Catskills, portions of the Green and Adirondack Mountains, Cresson, Pennsylvania, and Deer Park, Maryland, may also be mentioned as places which afford more or less complete relief to some. For further details as to this point the reader is referred to the works of Wyman and Beard.

Much attention has been devoted of late years, especially by laryngologists, to the eradication of the liability to attacks of hay-fever by means of the energetic local treatment of abnormal conditions of the upper air-passages, and notably of the nose. Some, indeed, cauterize noses which are, as far as the eye can see, perfectly healthy, with the aim of destroying or profoundly modifying the terminal filaments of the sensitive nerves, and thus, as it were, preventing the presence of irritants from being reported to the nerve-centres. The degree of success which has thus far been attained is certainly sufficient to make it desirable to treat thoroughly any local lesions which may be present; and the precious qualities of cocaine remove in large measure the difficulties which formerly lay in the way of the use of the galvano-cautery, chromic acid, and similar agents, even by a practised hand, and in children more than in adults. Dr. John N. Mackenzie tells the writer that, speaking generally, he considers the prognosis to be better in children than in grown persons; Dr. F. I. Knight, that in adults he has found the early more amenable to local treatment than the late forms. For all details as to methods the reader is referred to the appropriate portions of this work and to the standard writers on diseases of the throat and nose.

Kinnear reports a cure as following the use of the spinal ice-bag in several cases under his care, one of these a boy of about twelve. It is a severe remedy for a child, and also for those who have the care of it, to seclude it in closed and darkened rooms during the greater portion of the day; a plan which, however, mitigates the severity of the attacks. Wyman recommends that the windows of the sleeping-room be closed early in the afternoon and kept closed during the night. Motion of the air is thus avoided and an opportunity is given the dust to settle. The diet should be nourishing; flannel should be worn next the skin; and occasional warm baths with daily dry friction suit most persons better than cold bathing. The application of a solution of cocaine to the mucous membrane of the nose gives some temporary relief; but caution must be exercised, particularly in children, in the free use of this remedy.

There is no one drug which proves useful to so many cases as quinine. It should be given in full doses thrice daily, beginning two weeks before the expected outbreak and continued till near the close. Arsenic is another remedy which is of service to some, and is, as a rule, well borne by children: it should also be begun some time before the attack.

The list of drugs and of combinations of drugs for internal and local use which might be given here is a long one,—so long as to prove that none are very satisfactory or helpful to many persons. It is to be hoped that in the near future some plan of efficient home treatment for this distressing and rebellious complaint may be discovered. One fact speaks volumes,—that physicians who are sufferers and who cannot absent themselves from home, as a rule, after having tried various methods of treatment resign themselves to their fate and make the best of it.

PHTHISIS.

By A. JACOBI, M.D.

It was but a few years ago that the question could be raised in earnest whether tuberculosis and phthisis were identical. As great an authority as Ruehle denied that identity, though he admitted that phthisis was more than a mere inflammation, and questioned, though phthisis caused tuberculosis, whether the latter gave rise to the former in every instance.

Of late, not only are tuberculosis of the lungs and phthisis considered identical, but both are assumed to be the exclusive result of the invasion of a specific bacillus, whose effect consists in local irritation, with formation of small neoplasms and a morbid process with either an acute or a chronic course, the latter of which terminates in either extensive destruction or induration of tissue.

Its symptoms either are those of a general morbid condition, such as emaciation, pallor, fever, anorexia, perspiration; or there are some direct symptoms, such as cough, expectoration, dyspnœa, pain, and palpitation. Besides these symptoms, there is not infrequently the same invasion of a specific bacillus into glands, bones, and joints.

In the adult the tubercular deposits in the lungs prefer the apices. The reasons for this predilection are various. The lungs are firmly fixed at the hilus; thus the diaphragm cannot change the consistency of the pulmonary tissue and the lumen of the bronchial tubes to the same extent in the apices as in the lower lobes. Besides, the weight of the arms presses mostly upon the upper lobes. Furthermore, the current of air brought up from the lower part of the lungs is liable to repel the secretion trying to find its way out, into the upper lobes. This very secretion, the apices being less supplied with blood than the rest of the lungs, is thicker and more viscid, and prevents the air from getting in to the same degree as in the other parts of the lungs; and, finally, what has been called the phthisical habitus is mostly developed in the upper part of the chest, thus compressing the upper lobes of the lungs more than the rest. Thus the circulation in that part of the lungs is more sluggish, and bacilli which have once entered are not apt to be easily expelled.

Contrary to what we see in adults, in whom tubercular deposits mostly take place in the apices, the principal changes in the tuberculosis of children are often seen in the lower lobes. The reason may be found in the fact that the influence of the phthisical habitus develops in advanced years only. For the disproportion between the costal cartilages and the ribs, particularly in those cases in which premature ossification takes place, increases from year to year, thus adding to the difficulty of aeration in the upper part of the chest in the course of advancing years. Besides, the frequent attacks of broncho-pneumonia, which are apt to be the starting-points of tuberculosis, are more frequently observed in the lower lobes, and near the mediastinum.

Age.—According to Portal, tuberculosis of the lungs may be congenital. James Clark found it frequently after the second year; Meessen rarely in the first year, somewhat more frequently in the second; Koranyi very seldom before the third or fourth year. Ruehle met with acute miliary tuberculosis in some instances during the first period of life, with pulmonary phthisis, not, however, before the first dentition; Trousseau very often in the first years of life; Papavoine only between the fourth and fifth years; and of Guersant's hospital patients one-eighth of all those in the second year were tubercular.

The large institutions of New York City afford few facilities for adding statistical material of this kind, because of the very small amount of hospital accommodations for such children and the incompleteness of the information to be derived therefrom. But every practitioner with ample means of observation meets with a great many cases of general miliary and likewise pulmonary tuberculosis. Demme had under hospital observation in the course of twenty years 36,148 cases, 1932 of which were of tuberculosis; 1580 of the latter were pulmonary. Biedert collected 8332 cases of tuberculosis, 6.4 per cent. of which were those of children. Within three years Fürst observed 4000 cases of children's diseases up to the fourteenth year of life. Of the 330 tubercular cases among them, 247 were pulmonary; one was two weeks old, one six, one seven, fifteen from two to three months, seventeen from three to six months, forty from six to twelve months, sixty-six from one to two years, eighty-two from two to four years, thirty-nine from four to six years, forty-six from six to ten years, and twenty-two from ten to fourteen years. Thus, according to Fürst and Demme, the largest number of cases was met with between the second and fourth years. According to Baginsky, eight per cent. of all cases of pulmonary tuberculosis are met with below the tenth year.[1]

Some more points connected with the question as to the age at which tuberculosis may be met with, the reader will find discussed in the essay on tuberculosis contained in this volume.

Causes.—The etiology of tuberculosis in general has been treated of so extensively in the paper on tuberculosis just alluded to that I may be permitted to refer to it for all particulars. It is worth while, however, to insist upon a few points.

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[1] Maximilian Herz, Ueber Lungentuberkulose im Kindesalter, Wien, 1888.

In children the pulmonary artery is relatively larger; thus the lungs are more succulent and liable to furnish a very fair resting-ground for the bacillus. Besides, in the early years of life the right heart is still predominating, with the same result.

The invasion of the bacillus which is not only the cause of phthisis, but also the principal source of broncho-pneumonia and caseous pneumonia, may take place by direct inspiration. In every instance it is the smallest bronchi that furnish the best resting-place. In these cases the bronchial tubes are found thickened at a very early period. The upper air-passages, nares, pharynx, and larynx, being cooler and more exposed to strong currents of air, have therefore fewer cases of local tuberculosis. Even before the discovery of the bacillus, the inhalation of sputum was proved to be the cause of tubercular infection by Tappeiner, who at that early time accused beds and clothing of transmitting the disease. Contagion is not only not prevented by the drying up of sputum, but, on the contrary, it appears that as long as it is moist it is not attended with any particular danger. When tuberculosis develops from cheesy degeneration, the first changes are found in the blood-vessels or in the lymph-ducts and glands. The former are thickened, the latter enlarged.

Hereditary disposition has formerly been characterized from two points of view. A direct transmission can be proved in but few instances, but the propagation of a peculiar debility or inefficiency of either the whole organism or special organs deprives the individual of its power to resist injurious influences or deleterious invasions. Altogether, the number of cases in which hereditary influences can be traced is very great; in Demme's cases of tuberculosis of bones and joints in 69.6 per cent., in that of the lymphatic glands in 65.4, in visceral tuberculosis in 71.8, and in lupus in 37.8 per cent.

The relation of scrofula to tuberculosis has been amply discussed by Dr. Ashby in this volume. He proves that the assumption of a disposition on the part of scrofulous persons to become tubercular has to give way to the knowledge that what was called scrofulous was tubercular in many instances. In "scrofulous" deposits the bacillus tuberculosis has been found, and scrofulous material has been inoculated so successfully as to produce tuberculosis. Schüller inoculated caseous masses taken from a gland, with the result of producing tuberculosis of the osseous tissue; the same experiments of many observers resulted in general tuberculosis. Cohnheim proved the tubercular nature of fungous arthritis, caseous adenitis, and pneumonia; Cornil, of many hypertrophied glands and fungous synovitis; Demme, of ostitis, multiple periostitis, and the granulating ostitis of the phalanges. Many cases of chronic "scrofulous" eczema and nasal and aural catarrh exhibit the bacillus. Still, there are cases in which the latter is absent, but the necrobiosis (Virchow) of the glands is such as to facilitate the invasion of the bacillus and to impair the resisting power of the cells.

The introduction of the tubercular virus through the digestive tract, by means of the milk and meat of tubercular cows, particularly in cases of tubercular mastitis, is of at least occasional occurrence. It cannot be denied, though many feeding-experiments proved failures. Skin, mucous membranes, and glands are also ready gates for the entrance of the bacillus. It has been stated before that eczema and impetigo, scrofulous inflammations and abscesses, and nasal and aural catarrhs are liable to be infected with the bacillus. All these facts have been previously discussed by Dr. Ashby and myself.

The phthisical habitus may not give rise to pulmonary phthisis at all; a disposition is but one of the factors. Its definition comprehends a great many changes, not one of which, by itself, would appear dangerous. But the sum total of the symptoms exhibited even in early childhood has something very characteristic. There are the relatively great height of the body compared with its weight, the thin bones and muscles, transparent and delicate skin, scanty subcutaneous tissue, the extensive nets of superficial veins, the flushed or pale cheeks, pale mucous membranes, flat chest with short sterno-vertebral diameter, large intercostal spaces, shortness of costal cartilages either congenital or resulting from premature ossification, the marked depth of the supra- and intra-clavicular fossæ, the prominent scapulæ, the clubbed finger-ends, and the feeble heart.

Varieties of Pulmonary Tuberculosis.—Pulmonary tuberculosis is met with in three forms,—viz.: 1st, acute miliary tuberculosis of the lungs; 2d, acute or subacute caseous pneumonia; 3d, chronic phthisis.

Acute miliary tuberculosis has formerly been shown to result from the local tuberculosis of joints, bones, and glands. It is but the termination of the tuberculous process which, after having been local, becomes general through an extensive embolic distribution. Acute tuberculosis may also be mostly local, and death may set in before the disease becomes generalized. It is liable to remain confined to the lungs when the starting-point was from the bronchial or mediastinal glands.

Acute and subacute caseous pneumonia takes its origin from catarrhal (broncho-) pneumonia, as a rule; in some instances, from the fibrinous variety. It is attended with cough and fever (somewhat remitting in the morning), frequent and superficial respiration, all sorts of auscultatory signs, from the finest sibilant and subcrepitant to the large moist and dry râles, and occasional cyanosis, from a slight hue of the lips to the ashy discoloration of intense suffering. Bronchophony is more frequent than bronchial respiration. The results of percussion are not always conclusive; there are but slight changes sometimes: it is here that the gentlest tapping only will yield differences of sound. Recovery is apt to take place in from ten to fifteen days. Relapses—or, rather, new attacks—may occur, and still recovery take place. Particularly is this so in cases resulting from or complicated with pertussis or measles; they may last months. In many the respiration never becomes normal, either through induration of the pulmonary tissue, or through fatty degeneration or enlargement of the heart. Many such cases undergo extensive caseous degeneration,—mainly those which originated in whooping-cough, measles, scarlet fever, and diphtheria, particularly in such children as suffer from the results of rhachitic contraction and curvature, and incompetency of the thoracic muscles.

Chronic phthisis is the most frequent variety. Still, it is not common before the end of the first year. Fürst's cases[1] run from the fourteenth month to the twelfth year. But there is not a year which does not furnish me with a case or two at that early period. Children of a few years are frequently affected, and cases occurring at eight years and upward are by no means rare.

Their symptoms do not vary particularly from those of adults. In younger children some symptoms are difficult to discover. Cough is often overlooked for some time; it is short and apparently easy, or, on the other hand, hard, or loose, and mucous. Expectoration is either scanty, or is inaccessible to inspection and examination because it is swallowed. Hemorrhage, mild or severe, is of rare occurrence.

Temperature is high in the afternoon and in the night; remission takes place in the morning. But rarely the high temperature is met with in the morning. Sometimes the remission is so intense that the temperature becomes quite normal or even subnormal. Remission of too short a duration means danger. After midnight perspiration is as frequent and intense as it is in adults; it is liable to increase the tendency to emaciation, which is always very great. A girl of four years, weighing forty-five pounds, I have seen losing sixteen pounds in ten weeks. When, in addition, the digestion becomes disturbed and diarrhœa sets in, the fatal termination is reached sooner.

Respiration is superficial and frequent; this symptom sometimes precedes every other, before auscultation and percussion reveal anything. But in most cases there are one or more limited areas of dulness. Gentle percussion reveals it more readily than strong tapping. By itself, however, the dulness is no conclusive evidence of tubercular infiltration, for, as a result of simple interstitial inflammatory hyperplasia and cicatrization, retraction of pulmonary tissue, particularly below the clavicle, diminished respiration, prolonged expiration, even slight cavernous breathing resulting from dilatation of a bronchus, may remain behind. But in these old and permanent indurations the symptoms are not changeable, and there are no acute or recent ones to accompany them. In phthisis, however, there are auscultatory signs of an acute or a subacute character, and mostly quite extensive. Large and small rhonchi—viscid and loose, loud and fine, dry and moist, crepitant, subcrepitant, sibilant (particularly on deep inspiration) —are heard together or in alternation. Now and then there is bronchial respiration; still, bronchophony is much more frequent than bronchial respiration, because of the relative smallness of the infiltrations which permit of air-space between them; cavities yield cavernous breathing in proportion to the size of the abscess. When it is small, as it is apt to be, cavernous breathing is very apt to disappear temporarily, when the cavity fills up with secretion or pus.

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[1] Maximilian Herz, Ueber Lungentuberkulose im Kindesalter, Wien, 1888.

Pathological Anatomy.—In dead bodies the results of the tubercular process are various; slight they are but rarely. Indeed, I remember but a single case, that of a girl of six years, who died suddenly at a very early period of the disease, of hemorrhage. The post-mortem appearances differ in acute and chronic cases. In the former the tubercular deposits are gray, after some time yellow, small, and very numerous. A great number are found on the bronchioles, many of which are thickened. When the process lasts longer, infiltrations take the place of nodules, through confluence; the bronchial glands are swollen, sometimes cheesy in the centres, and the pleuræ are adherent.

The invasion of the bacillus results in local irritation and hyperæmia, emigration of leucocytes, formation of giant cells, and increase of the epithelial cells. Thus miliary nodules are formed and the connective tissue is increased; thus tubercular infiltration is brought on, and the lumen of the bronchus may become narrow, and atelectasis result therefrom. The tubercle, being without vessels, is apt to undergo caseous degeneration; thus the alveoles are filled with the caseous mass, and form small cavities, many of which coalesce by the disappearance of the perishable septa and develop into cavities of larger or even immense size. The transmission of the process into other parts of the lungs takes place either in the proximity, by contiguity of tissue, or through blood-vessels or lymph-ducts. Sometimes the formation of cavities takes place late, if at all; in such cases a whole lobe may be solidified, partly through large masses of tubercular infiltration and partly through the new formation of interstitial tissue. Its hyperplasia takes place through the proliferation of connective-tissue cells and their transformation. Its existence prolongs the course of the disease and affords a certain degree of safety; for not infrequently it forms hard and thick capsules for small or large abscesses, which thus are deprived of a great deal of their danger. They may even be retained so long that exsiccation and calcification occur.

Other anatomical changes are the following: bronchiectasis,—the bronchial tubes become dilated by the shrinking of the adjacent newly-formed connective tissue; emphysema in the pulmonary tissue not yet filled with tubercle; suppurative pleurisy, in consequence of the presence of tubercles in or near the surface of the pleura, and through the direct communication of blood- and lymph-vessels between lung and pleura, in which case adhesion and thickening of the pleuræ become additional causes of disturbances of circulation and blood-supply; pneumothorax, when the pleura was perforated before adhesion became established. Finally, dilatation of the right ventricle, often with fatty degeneration of the heart-muscle, is the frequent result of the difficulty encountered by the cavities of the heart in trying to discharge its contents.

Symptoms.—One of the earliest symptoms of pulmonary phthisis is atrophy in many of the patients. It is more common in the very young than in those of more advanced years. I knew a tubercular baby of seven months that weighed exactly seven pounds. This atrophy is probably so intense for the reason that the disease is not confined to a single organ. The skin is flabby, waxy, yellowish or white, wrinkled, inelastic, and often covered with pityriasis; the bones, cheeks, and scapulæ are prominent; the eyes half closed, or open and staring, without expression, listless. The subcutaneous tissue is very scanty, the voice thin, and the cry low or inaudible. These symptoms of complete atrophy, however, are not characteristic of tuberculosis; but in every case of atrophy the lungs ought to be examined with the utmost care, no matter whether there is much cough or not. Pulmonary changes may be very much advanced and still the physical symptoms not very evident, and, again, tubercular infiltration not very extensive but the physical signs very perceptible. Now and then those of catarrh or of pleurisy only can be found, both of which may improve either spontaneously or under treatment.

It is the totality of the symptoms that is important for diagnosis,—the simultaneous existence, for instance, of hereditary influence, chronic eczema or impetigo, disease of bones and joints, glandular swellings, some dyspnœa, cough more persistent than, perhaps, violent, and the permanence and relative invariability of the physical signs.

Fever is more distinctly noticed in children of somewhat advanced age. The temperature must be taken frequently, inasmuch as remission may be expected daily and the temperature is sometimes subnormal. The fever is either continuous or hectic, or its type is inverse. Brunniche found that the morning temperatures are apt to be higher than those of the evening in all cases in which pulmonary tubercular infiltration is complicated with miliary general tuberculosis.

Cough is not a prominent symptom in the incipient stage of chronic tubercular infiltration of the lungs. It is sometimes not noticed at all by the attendants, is frequently merely short and hacking like that arising from a slight pharyngeal irritation, and becomes more frequent and vehement later on. It may then often be found paroxysmal, resembling that of whooping-cough, with cyanosis, dyspnœa, and vomiting. It may be dry and very painful, the pain being attributed to the epigastrium, the muscles of which are under a perpetual strain; or moist. Still, sputa are scanty, for the expectoration is swallowed as soon as it reaches the pharynx. When some of it is obtained, the microscopical appearance is that found in more advanced age. Of pulmonary elements there are disintegrated alveolar epithelia and elastic fibres of lung-tissue. Bacilli are found, but not always so readily as in the adult. The method of their discovery is amply described by Dr. E. O. Shakespeare on page 165 of the first volume of this work.

Blood is not a frequent admixture in the expectoration of phthisical children. Now and then it is met with, but profuse hemorrhages are rare in children. They may be idiopathic, for in one case of I. Hoffnung's no disease of a lung could be found. One case of his occurred from thrombosis of the pulmonary artery, one from pulmonary apoplexy in a new-born child, two from gangrene, one from a suppurating gland which perforated into a branch of the pulmonary artery and a bronchus, and five in pulmonary phthisis. In four of the latter the bleeding came from a ruptured aneurism of the pulmonary artery. I do not remember more than half a dozen cases of pulmonary hemorrhage in children except those which took place in violent attacks of whooping-cough. Only one of my cases—phthisis—was three years old; one, a girl of eleven, had repeated attacks extending over a year, which appeared to depend on or were accompanied by a mitral insufficiency, and exhibited infiltrations of the upper lobe afterwards; the others occurred in children affected with phthisis, early or late stage, of from seven to eleven years. From a diagnostic point of view the absence or presence of larger amounts of pus may be noticed. I remember cases of pulmonary abscess, a few of them resulting from perforating empyema, which bled quite freely. In pertussis copious hemorrhages are frequent. They may become dangerous in this, that blood coagulating in the finest bronchioles may give rise to local collapse of the lung—atelectasis—and lobular pneumonia in consequence, in this way increasing the disposition or liability to tubercular invasion.

The part played by the lymphatic glands is a very important one. Their primary swelling may be due to general "scrofulosis," or result from the bronchial catarrhs so often met with in small children, particularly those affected with rhachitis and pertussis. The disintegration and liquefaction of their centres may give rise to embolic processes and result in pyæmia. The mucous membrane of the respiratory surface being hyperæmic and eroded, the bacillus finds its way into the gland, where it irritates and produces the changes mentioned above. Two possibilities then arise. The bacillus may not stop long in the gland, but may be carried through the vasa efferentia into the circulation, and thus light up a miliary tuberculosis. Particularly is this the case where the gland is in close communication with large lymph-channels; thus peritoneal tubercles are very apt to be carried into the thoracic duct. Or the irritation produced by the presence of the bacillus can give rise to excessive formation of connective tissue; the capsule of the gland and its interstitial tissue will be thickened, and thus the bacillus locked up. Thus the gland may reach a considerable size, and feel fairly hard to the touch, even when its centre is already much advanced in its softening process. The very size of the glands may give rise to serious symptoms: the circulation of the pulmonary artery and vein, the superior cava, and the jugular may be compressed, resulting in œdema, hæmoptysis, infarctus, and considerable swelling of external veins, very probably, also, in passive accumulation of blood in the cavities and the muscle of the heart. Their softening and suppurative perforation affect, and infect, the neighboring parts of the lungs. Thus it is that the tuberculous process is so very apt to begin, and to be most extensive, about the hilus, where the glands are present in large numbers. The pneumogastric nerve, too, and its branches, are annoyed by numerous and swelled bronchial glands. Persistent hoarseness, before any laryngeal symptoms can be made out, and indeed before those of phthisis have been developed at all, can be explained in this way. Fleischmann observed a case of intense laryngo-spasm which was thus caused. Early pleuritis, and dull pain posteriorly, here find their explanation. Intense dyspnœa may be the result of large glandular swellings and their mechanical effect upon a large bronchus or the trachea, and hæmoptysis that of a glandular abscess perforating into a blood-vessel. All such occurrences may take place unexpectedly. For the presence of large masses of glandular swellings is not easily diagnosticated, sometimes is not even suspected. The closed cavity of the chest does not permit palpation, auscultation is sometimes not successful because the respiratory murmurs are easily transmitted through the solid bodies, and even percussion does not always give a satisfactory result. But quite often the local absence, or diminution, of respiration, or the coarse character of the latter in a limited locality, besides dulness over the manubrium sterni, and occasionally near its left or right margin, together with the presence of glands about the neck, in the axilla, and in the inguinal regions, bids fair to facilitate a correct diagnosis.

Complications.—The complications of pulmonary tuberculosis with tuberculosis of other organs are very frequent. I hardly remember a case of the former without an affection of the pleura, either simple adherent, or suppurative, or tubercular pleuritis, or pneumothorax. Tubercular meningitis is not frequent in cases of chronic phthisis, but in those complicated, either from the beginning or towards the fatal termination, with miliary tuberculosis of the lungs, it is often found as the result of the distribution of the process over the whole system. The liver participates with a perihepatitis which sometimes glues the organ to the diaphragm, or with fatty degeneration, which is quite common in chronic phthisis, or with small or large tubercular deposits upon or in the liver. Their size varies: some are large, the majority small. They undergo softening but rarely. The tubercular degeneration of the system is of a similar nature, perisplenitis and tubercles being met with, but not so commonly as in the liver. The kidneys exhibit the same class of changes, only in smaller numbers. Pyelitis has been observed as the result of the disintegration of a tubercle, and abscesses in the parenchyma I have seen myself, from the same cause. The stomach suffers less than most other organs. Externally tubercles are found as a part of tubercular peritonitis, internally an ulceration has been found occasionally; its functions are often not disturbed. Gastric catarrh may result from the impediment to circulation connected with every pulmonary or cardiac disease, but, as a rule, the function and particularly the secretions remain normal, and facilitate the ingestion and assimilation of large quantities of food. The bowels participate much more freely. In a chronic consumption they are rarely normal; hyperæmia is frequent, and ulcerations are not uncommon. They are mostly found in the lower parts of the small intestine, as future papers will show, but not uncommonly also in the duodenum, cæcum, colon, and in protracted cases even in the rectum.

Prognosis.—The prognosis depends on a great many factors. Intense scrofulous diathesis and hereditary disposition, and protracted morbid processes in glands, bones, and joints, yield a bad prognosis, though the duration of the tubercular process be ever so long. Measles and whooping-cough contracted under such circumstances are bad, because they are liable to lead to extensive lesions of the lungs. They occur frequently between the second and the fourth year, and therefore tuberculosis is readily developed at that age. Those cases which occur in the first year, as also those before puberty, about and after the tenth year, are quite unfavorable. Rapid increase of atrophy, with loss of appetite, is bad. So are rapid respiration and persistent high temperature, cyanotic hue and night-sweats, and the presence of a cavity. The permanence of mixed auscultatory symptoms, such as fine sibilant and moist râles, large moist rhonchi, and bronchial respiration (or only bronchophony), is a very ominous sign.

Treatment.[1]—Hereditary predisposition to tuberculosis being quite frequent, and transmitted even by parents who still appear to be in fair health, every catarrh in the children of such parents must be carefully watched. The premature ossification of the costal cartilages, most frequently found about the superior part of the chest, and the consecutive shortening of the sterno-vertebral diameter, give rise to contraction of the thorax and insufficient expansibility of the (upper lobes of the) lungs. In such cases the aeration of the blood suffers at a very early date, catarrhal and inflammatory thoracic diseases are liable to become dangerous, and gymnastic exercises are required in early childhood.

Direct transmission from the parents to the children is probably not frequent, but it is possible, and therefore the child must not share the room and bed of the consumptive. Kissing must be refrained from; it may often be the cause of contagion, though tuberculosis is not so frequently transmitted in that manner as some other diseases,—for example, diphtheria.

A consumptive mother must not nurse her infant. She is a greater danger than one afflicted with syphilis. Her milk is a positive injury, as is the milk of tubercular cows, though the udder may not be diseased. Two cows out of a hundred are tubercular. Hence the least that can be done is to boil the milk intended for the nourishment of the infant. By thus observing the rule which I have enjoined these twenty-five or thirty years, the milk can be made more innocuous than is possible for the butter or cheese obtained from such cows. These rules ought to be strictly obeyed, though there be exceptions to the universal experience. An instance of such exceptions is mentioned by Biedert, than whom there is no more reliable observer. He reports the cases of children who were fed a long time on the milk of tubercular cows without being attacked themselves.

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[1] Some of the following pages are from the Archives of Pediatrics, October, 1888.

Great care must be taken in the selection of a wet-nurse, and of the help about the house with whom the children are to be in contact. The air about the house and about the school must be pure, the school-hours interrupted by physical exercise, and chronic ailments, such as rhachitis, carefully watched and treated, to avoid the debility of the tissue which facilitates the invasion of the tubercular guest. It is particularly measles and whooping-cough that must be carefully watched.

But all these and other measures which are the results of the different adjuvant causes in the development of tuberculosis have been elaborately discussed in my paper on tuberculosis, to which I here refer.

Among the causes of consumption monotony of food has been enumerated by many. It is evident that it cannot account for much in the cases of infants or children, whose habits are plainer and their digestive functions more adapted to simpler and more uniform articles of diet. Most of these, while in health, are satisfied with milk, cereals, and but little meat. Sweet cream may be added to the milk, but more than a few ounces are not digested through the course of a day. Cod-liver oil acts mostly through its fat. During the afebrile condition and chronic emaciation of phthisis, over-alimentation, introduced by Debove, may be tried to advantage, while insufficiency of gastric digestion, if it exist at all, may be stimulated by the administration of artificial gastric juice (pepsin with muriatic acid) and mild stomachics (gentian, nux, diluted alcoholic beverages). Where exercise cannot be procured to a sufficient extent, or is contra-indicated by the necessity of enforcing temporary, but absolute, rest, massage, according to S. Weir Mitchell's plan, will take its place. During fever, over-alimentation has to be stopped; it deranges digestion and slowly increases the fever. Alcoholic stimulants will at that time often take its place to advantage. While they do not act well in certain over-irritable natures, with over-sensitive hearts, and in hæmoptysis, they are good stimuli for the general system, diminish perspiration, and act favorably in diarrhœa.

In the treatment of tuberculosis no single factor is beneficial by itself. The quality of the air alone will not cure the sick, any more than will a certain mixture of salts and water in a mineral spring, or some known chemical relation of albuminoids and carbo-hydrates in an article of food. Insufficient clothing and bedding, unheated rooms, draughty halls, indigestible food, strong coffee and tea, hot cakes and cold drinks, late hours, lively hops, brass instruments and pianos disturbing midnight rest, kill as many, in proportion, in Colorado, Florida, Southern France, and Italy, as in New York. It must never be forgotten that the change of climate is mostly a negative remedy, and cannot be expected to offer more than the possibility of favorable external circumstances.

Moist air is a better conductor of warmth than dry air. Hence loss of temperature is more rapid in moist air than in dry air. Dry air, therefore, may be very much cooler, and is still better tolerated in spite of its lower temperature, and affords more protection. In adults hæmoptysis appears to be a frequent occurrence during the season of increasing atmospheric moisture (spring). According to Rohden's researches, a rapid increase of the percentage of water in the blood is frequently sufficient to produce a hemorrhage. The drinking of large quantities of water, therefore, ought to be avoided, and no residence should be selected for a patient subject to hæmoptysis where the atmosphere is very moist. Dry altitudes, such as those of New Mexico, have given me good results in pulmonary hemorrhage. At all events, no place must be selected where the percentages of moisture in the air are liable to change rapidly. The uniformity of an insular climate, while benefiting the average case of phthisis, is, therefore, not so dangerous to those who have bled from their lungs. Nevertheless, dry air and a higher scale of the barometer are preferable.

The diversity of opinions in reference to the climato-therapeutics of phthisis resulted from the circumstance that the indications were not distinctly understood. Neither cold nor warm, neither dry nor moist, air by itself is a remedy. Warm air does not cure, but it enables the patient to remain out of doors. The temperature must be uniform, sudden currents of air avoided, and the atmosphere free of microphytes. At an altitude of sixteen hundred feet their number is greatly reduced (Miquel), there are but few at a height of two thousand six hundred feet (Freudenreich), very few at six thousand, and absolutely none at twelve thousand feet, provided the parts are not, or but little, inhabited. Over-population of elevated villages and cities diminishes or destroys their immunity. In the factories of the Jura Mountains, with a large working population, at an altitude of three thousand five hundred feet, tuberculosis is frequent.

Protection against sudden gusts of wind and rapid changes of temperature is an absolute necessity. The elevated valleys (or rather recesses of mountains) of Colorado deserve their reputation in pulmonary diseases. Davos is dusty, windy, and exposed to frequent changes of temperature during the summer, and must not be advised for that season. Woods are warmer in winter, cooler in summer; so is the ocean. Both, therefore, well deserve their reputation in the chronic ailments of the respiratory organs.

Not the thinness of the atmosphere, but its purity, is the requisite, together with a high percentage of ozone. The latter is developed under the influence of intense light, the presence of luxuriant vegetable growth, particularly of evergreen trees (Terebinthinaceæ), and the evaporation of large sheets of water. Thus, ozone is found on moderate or high altitudes, in needle-wood forests, and near or on the ocean.

In the general hygienic treatment of tuberculosis the skin requires particular attention. Sudden changes of temperature, which strike the surface suddenly and work their effects on internal organs by reflex,—"colds,"—

in spite of the modern superciliousness of some who deny any pathological change unless the exclusive work of bacteria, will always hold their places in nosology. The skin must be both protected and hardened. Wool, or wool and cotton, must be worn near the skin, the feet particularly kept warm, no wet or moist feet permitted, undergarments changed according to season and the alternating temperatures of days or weeks, and every night and morning. It is of the greatest importance to impress upon the minds of the very poorest that they must not wear during the day what they have slept in. Still, while protection is to be sedulously sought after, vigor is to be obtained by accustoming the surface to cold water. The daily morning wash may be warm at first, and become gradually cooler,—alcohol being added to the water in the beginning (alcohol alone is unpleasant through its withdrawing water from the tissues), and salt always. The temperature of the water being gradually diminished, the same treatment can be continued during the winter, with a pleasant sensation of vigor. The subsequent friction with coarse bathing-towels sends a glow over the surface and through the whole body. The easiest way to start the habit is by washing; a short sponge- or shower-bath will take its place soon, and a cold plunge will be borne even by the weak afterwards.

It has become fashionable with many to feign a contempt for internal medicines in the treatment of tuberculosis, pulmonary and otherwise. I am glad I cannot share their opinions. Thus, for instance, I look upon arsenic as a powerful remedy in phthisis. It was eulogized as early as 1867 by Isnard, in a monograph, for its effect in both malaria and consumption, in both of which he explained its usefulness through its operation upon the nervous system. He claimed that suppuration, debility, emaciation, vomiting, diarrhœa, and constipation would improve or disappear under its administration. The doses of arsenious acid used by him in the cases of adults amounted to from one to five centigrammes (one-sixth to five-sixths of a grain) daily.

Arsenic is certainly a powerful remedy. It is known to act as a poison and a strong caustic. It prevents putrefaction, though as an antiseptic it ranks even below salicylic acid. It acts favorably in malaria, chronic skin-diseases, and maladies of the nervous system, and has considerable, and sometimes unexpected, effects in the treatment of lympho-sarcoma and sarcoma. It is also said to improve, in the adult, sexual desire and power, and in animals physical courage. Thus there is a variety of effects the intrinsic nature of which may be found, uniformly, in the action of the drug on the function and structure of the cell, which, though varying in different organs, has the same nutritive processes. Arsenic has a stimulating effect on cell-growth. In small and frequent doses it stimulates the development of connective tissue in the stomach, in the bone and periosteum, everywhere; in large doses, by over-irritation, it leads to granular degeneration. Like phosphorus, arsenic builds in small doses, destroys in large ones. By fortifying the cellular and all other tissues, both fibres and cells, it enables them to resist the attack of invasion, both chemical and parasitic, or to encyst or eliminate such enemies as have penetrated them already. Thus it finds its principal indication in the peculiar fragility of the blood-vessel walls resulting in pulmonary hemorrhage.

The doses must be small. A child a few years old may take two drops of Fowler's solution daily, or a fiftieth or fortieth of a grain of arsenious acid, for weeks or months in succession. This amount may be divided in three doses, administered after meals, the solution largely diluted. There is no objection to combining it, according to necessity, with stimulants, roborants, or narcotics, and to giving it for an indefinite period, unless the well-known symptoms of an overdose—gastric and intestinal irritation and local œdema—make their appearance. But they seldom will, particularly when small doses of opiates are judiciously added to it. In almost every case, perhaps in every one, it is desirable to administer it in conjunction with digitalis.

In the vertebrate animal digitalis increases the energy of the heart-muscle and its contraction; thereby it increases arterial pressure and diminishes the frequency of the pulse. By increasing arterial pressure it favors the secretion of the kidneys, improves the pulmonary circulation, empties the veins, thereby accelerates the flow of lymph and the tissue-fluids, and exerts a powerful influence on the metamorphosis of organic material,—that is, general nutrition. In addition, what it does for the general circulation and nutrition it also accomplishes for the heart-muscle itself. The blood-vessels and lymph-circulation of the latter are benefited equally with the rest. Thus digitalis, while called a cardiac stimulant, contributes largely to the permanent nutrition and development of the organ. This effect is not only of vital importance for the economy of the system on general principles, but an urgent necessity in view of the fact that there appears to be a relative undersize of the heart, either congenital or acquired, in cases of phthisis; and there is certainly such a predominance of the size of the pulmonary artery in the young, particularly over the aorta, that the normal succulence of the lung becomes pathological quite readily when the insufficiency of the heart-muscle tends to increase low arterial pressure within the distributions of the pulmonary. The selection of the preparation to be administered is not an indifferent matter. The infusion and the tincture are sometimes not well tolerated by the stomach; digitalin, not being a soluble alkaloid, but a glucoside, is not always reliable in its effects, nor of equal consistency and strength; a good fluid extract, or the extract, is borne well and may be taken a long time. A child a few years old may take about two minims of the former daily, more or less, for weeks and months, or its equivalent in the shape of the extract (two-thirds of a grain daily); the latter can easily be given in pills, to be taken in bread, or jelly, and combined with any medicines indicated for special purposes, such as narcotics, or nux, or arsenic, or iron,—the latter to be excluded in all feverish cases, or in all cases while fever is present. So long as there is no urgent necessity for a speedy effect, digitalis will suffice by itself; as a rule, it does not operate immediately in the small doses above mentioned. The addition of strophanthus, or spartein, or caffein, all of which are speedily absorbed and eliminated and exhibit their effect rapidly and without the danger or inconvenience of cumulation, will prove advantageous in many cases.

Other medicines have been used in great numbers. Specifics have been recommended, and symptomatic treatment has been resorted to. The success of the latter depends on the judgment of the individual practitioner. No text-book or essay can teach more than general principles and their adaptation to the average case, and the measures to be taken in a number of exceptional occurrences. The indications for the use of narcotics, stimulants, expectorants, and febrifuges will change according to the cases and their various phases and changes. In every case the necessity may arise for antipyrin, antifebrin, phenacetin, salicylate of sodium, or quinine. It may be necessary to decide the question whether the administration is to be made through the mouth, rectum, or subcutaneous tissue, or how their effects are to be corrected or combined. I have often found that a hectic fever would not be influenced by quinine, or by antipyrin, or by salicylate of sodium, but the combination of the first with one of the latter would frequently have a happy effect. However, in a great many cases where the fever persists, the use of quinine in sufficient doses, from five to ten grains daily, proves more satisfactory than the modern antipyretics with their prompt but temporary action.

The change in our pathological views, or rather the addition of a new factor in our etiological knowledge, has directed our attention to the antisepsis of the respiratory organs. It is not necessary to destroy bacteria in order to make them relatively harmless. It is impossible to kill the bacillus without killing the normal cell, but very mild antiseptics suffice to stop the efficiency and proliferation of the parasite. Thus we can hope that the future will teach us to reach the destructive process in the lungs. It is quite possible that the inhalation of hydrofluoric acid will not prove more beneficial than the rectal injection of sulphide of hydrogen, but the internal use of creasote (one to three minims to a child daily) and terebene (two to four minims every two or three hours) and the inhalations of turpentine, eucalyptol, menthol, and many others, appear to rouse our hopes for a future effective treatment. Much more than hopes we cannot have at this moment. But it is useless to despair, either passively or actively. For the present, it is certainly a desperate activity which tempts an enterprising hero of the reckless knife to cut away a part of a lung which is the seat of a general and disseminated process, and a misdirected enthusiasm tempered by mercenary tendencies that pretends to bake bacilli out of existence by means of a clumsy and inefficient apparatus.

Ulcerations of the tongue and pharynx are painful sometimes to such an extent as to require frequent attention. A well-directed spray of one part of nitrate of silver in two hundred parts of distilled water (glass to be of neutral, blue, or black color), administered once a day, will be found serviceable in average cases. Some are so bad as to interfere seriously with deglutition. I have been obliged to use a cocaine spray before every meal.

Gastric catarrh must be relieved, for a healthy stomach is indispensable for the economy of the organism. It is liable to suffer from the disordered pulmonary circulation, but just as often suffers by mistakes made in the diet of the patient. Large quantities of alcoholic beverages or the same not sufficiently diluted are often the causes of disturbances. So is iron which has been given injudiciously for the alleged purpose of meeting the prevailing anæmia. Milk is sometimes not tolerated; it may be substituted by buttermilk, koumys, kéfir, matzoon, or peptonized milk; or it may be prepared with dilute hydrochloric acid, in such a manner that one part of the latter is mixed with two hundred and fifty parts of water and five hundred parts of raw milk; the mixture is then scalded: it keeps better than plain milk, and proves very digestible. Or milk may be mixed with barley, oatmeal, rice, etc., or replaced altogether, temporarily, by farinaceous food. Fermentation in the stomach requires resorcin, bismuth, or creasote; the anorexia of intense chlorosis is sometimes benefited by small doses of sulphur; and a protracted catarrhal condition may be speedily improved by the washing out of the stomach with warm water in which bicarbonate of sodium, resorcin, or thymol in small doses has been dissolved.

As tubercular patients are liable to be affected with pleural irritation and inflammation, they must not undergo great exertions, as climbing, or give way to boisterous laughter. An attack of pleurisy requires a recumbent posture, mostly in bed, and warm poultices. A subcutaneous injection of a small dose of morphine will relieve the pain, and table-salt, half a teaspoonful to a teaspoonful in water, several times a day, proves the best of diuretics and absorbents.

Among the localizations of tuberculosis in children, that of the larynx is not frequent, but it is met with. According to Heinze, laryngeal tuberculosis is not produced by contact, but through the medium of the blood. But the expectorated masses are undoubtedly a frequent cause of the local infection, and, as a rule, the larynx is invaded rather than the lungs. Besides nodulated inflammatory swellings in the mucous membrane, submucous tissue, and glands, sometimes even between the muscles, there are small granulations and ulcerations on the cords, with universal catarrh, œdema, and phlegmonous destruction. The symptoms are those of catarrh and ulceration, and depend on the locality and severity of the lesion. In some cases the diagnosis of pulmonary tuberculosis could not be made in the beginning, and that of the local affection was based on the duration of the ailment, the persistence of the fever, and the steady emaciation. At first the laryngoscopic examination revealed catarrh only, and later ulceration and infiltration. The local treatment is that of the catarrh,—inhalation of warm vapors, steam, turpentine, carbolic acid, muriate of ammonium; poultices round the neck; opiates at bedtime. The lactic-acid spray and the application of iodoform have not served me so well as a daily spray of a solution of one part of nitrate of silver in from two to five hundred parts of distilled water. Stronger solutions are rather harmful. The pain produced by ulcerations located on the epiglottis and arytenoid cartilages is somewhat relieved by the application (by brush or spray) of bromide of potassium, morphine, or cocaine, or an appropriate mixture of two or three of them.

The air around patients suffering from laryngeal phthisis may be moist; but it is a mistake to believe that it must be warm. Cold air is warmed before it enters the larynx and lungs, provided it enters the respiratory tract through the nares. Only when it is admitted through the mouth does it remain somewhat cool when reaching the larynx. Thus the nares must be kept as normal as possible, and competent no matter with what difficulties. Nor will open windows interfere with the comfort of the patient, provided draught is avoided: this can be easily accomplished by screens or otherwise.

Tubercular ulcerations of the intestines may descend to the rectum; in that case the local symptoms, and mainly the tenesmus, may be alleviated by warm injections containing gum acacia or bismuth, with or without opiates. Food and drink must be warm. Bismuth may be given in doses of from two to ten grains every hour or two, so as to form a protection to the sore intestine. Tannin I have not seen do very much good. Naphthalin sweeps the whole length of the tract and acts favorably as a disinfectant. I have seen almost immediate improvement after its use. From four to ten grains may be given daily. Now and then the stomach rebels against it; in that case, resorcin, in doses of from one-fourth to one grain, in powder or in solution, may be given for the purpose of disinfection from three to eight times. Though it is very soluble, it is effective to a certain extent. All of the above may be combined with bismuth, or lead, or opium. Such preparations of salicylate of bismuth as were accessible have not rendered the services I had expected to obtain, judging from the reports of some European writers. Hydrargyrum bichloride cannot be relied upon for any effect in the lowest parts of the intestinal tract, because of its great solubility, the necessity of great dilution, and its ready absorbability.

Fistula in ano is a rare occurrence in children under all circumstances. I remember but two cases, in tuberculous girls of about ten years. No matter whether they be accidental complications, or the tubercular poison (bacilli) be conveyed to the parts through the circulation, or the fistula be the result of the presence, in the fæces, of bacilli, and their action on defective epithelium, practice has changed entirely during the last decade. The axiom that fistulæ in a consumptive patient must not be interfered with has given way to a more rational theory and sounder practice. The sooner they are operated upon and treated, the better.

In pulmonary hemorrhages the application of a lump of ice or an ice-bladder over the locality of the hemorrhage acts favorably, either through the direct influence of the cold temperature or through the reflex contraction of the bleeding vessels. Subcutaneous injections of fluid extract of ergot, or of ergotin in glycerin and water, are apt to give rise to induration or abscesses: hence it will be left to the practitioner to decide in an individual case whether that risk may be taken. Sclerotinic acid has been recommended for the same purpose. A syringeful has been injected hourly of a solution of one part in five of water. It is claimed that no local injury is done by it; but it is painful, and has been corrected by the addition of morphine. The latter may be given internally also for the purpose of relieving the patient's symptoms, both objective and subjective. If it cannot be swallowed well, the proper quantity of Magendie's solution, not diluted in water, is readily absorbed through the mucous membrane of the mouth or throat. The internal administration of ergot may be supported by that of mineral acids and digitalis. Of the latter, a single dose of from two to five grains, or its equivalent, acts well. The dilute sulphuric acid is both efficient and palatable; ten or fifteen drops in a tumbler of (sweetened) water will be readily taken to advantage. Acetate of lead, in doses of one-sixth to one-half of a grain, every hour or two, according to age and the severity of the case, is preferable to tannin; it can be given with morphine or digitalis, or both. The patient requires absolute rest and encouragement, and must be induced to make long forcible inhalations, and told to suppress the cough as much as possible. To relieve it opiates may be required. For the purpose of stopping hemorrhages the inhalation of the sesquichloride of iron (one to one hundred) has been recommended. As it was not expected to enter the bronchial tubes, its effect was presumed to be by reflex action. I have tried it, but cannot sufficiently recommend it.

As a general rule, among adults as well, a subcutaneous injection of morphine in the very beginning has a good effect. The pulse becomes fuller and softer, the patient quiet. The application of a ligature round an extremity I have not had occasion to try on a child. So long as there is any bloody expectoration the patient must remain in bed, and be kept on plain and fluid food.

Night-sweats are not uncommon in the tubercular phthisis of children from five to twelve years of age. They are favorably influenced by the same remedies which are apt to relieve the adult; such are sponging with vinegar and water, or alum, vinegar, and water. A powder of salicylic acid three parts, oxide of zinc ten, and amylum ninety, or salicylic acid three, amylum ten to twenty, and talcum eighty or ninety, dusted over the suffering surface, is quite beneficial and soothing. For internal administration the dilute sulphuric acid, ten or fifteen drops in a tumblerful of water, is found enjoyable by a great many. A single dose of atropinæ sulphas (one-three-hundredth to one-hundredth of a grain) at bedtime, or agaricin (one-fiftieth to one-twentieth of a grain), or duboisin (one-hundredth to one-fiftieth of a grain) will succeed in bringing relief. When there is an indication for opium, it may be combined with any of them. When the digestion is good, a sufficient dose of quinine (three to six grains), with or without ext. ergot. (the same dose), or ext. ergot. fluid. (one scruple to half a drachm), deserves a trial when for any reason the above remedies are discarded.

PLEURISY.

By E. N. WHITTIER, M.D.,

AND

H. F. VICKERY, M.D.

Definition.—An inflammation of one or both pleural surfaces, acute or chronic, primary or secondary, circumscribed or general,—essentially the same as inflammation of other serous surfaces, but more frequent.

Synonymes.—Pleuritis (*πλευρόν*, a "rib"), Morbus lateralis; French, Pleurésie; German, Pleuritis, Rippenfellentzündung, Brustfellentzündung.

History.—Hippocrates (460–357 B.C.) wrote of pleurisy (*πλευρίτις*), but without differentiating it from pneumonia. Later writers contended for the existence of a separate inflammation of the pleura; but it was not until about the beginning of the present century that Pinel vindicated this claim by demonstrating the morbid anatomy of the disease. Clinically, the discovery of auscultation by Avenbrugger (1761), and of the stethoscope by Laennec (1815), enabled physicians for the first time to make accurate and reliable differential diagnoses of pleural as well as of all other thoracic affections.

A few words as to the development of thoracentesis are indispensable. From the time of Hippocrates, various surgical methods of removing liquid pleural effusions were employed, most frequently incision. Over two hundred years ago, Scultetus advocated puncture and aspiration. Trousseau (1840) exerted a powerful influence in favor of the employment of the trocar and canula. In 1850, Dr. Morrill Wyman, of Cambridge, Massachusetts, revived aspiration; and his idea was at once appreciated and gladly adopted by Bowditch, of Boston, whose teaching and example have won for the operation universal acceptance.

A knowledge of the prophylactic value of asepsis in this as in all other surgical measures is of course due to Listerism.

Etiology.—Pleurisy is more frequent in spring and winter than in summer and autumn. Boys are somewhat more liable to the disease than girls. Of six hundred and sixty-two cases of death from pleurisy under the age of sixteen, Oesterlen states that three hundred and fifty-nine, or fifty-four per cent., were boys. In adults, five men have the disease for every three women. It is less frequent in children than in adults; but the marked disproportion once thought to exist has vanished with increased ability to detect its physical signs in the young.

Primary pleurisy, as we have seen to be true of the disease in general, is most common in cold, changeable weather. It attacks the poor and feeble by preference. That it is usually due to "taking cold" is maintained by many, but denied by others. Ziemssen, out of fifty-four cases of primary pleurisy, did not ascribe one to cold. Conclusive statistics on this point are wanting. The primary form is slightly more frequent on the left than on the right side.

As causes of secondary pleurisy are to be reckoned: traumatism (sometimes even a bruise); pulmonary disease,—pneumonia (catarrhal or croupous), tubercle, hemorrhagic infarction, abscess, gangrene, and tumors; hemorrhage into the pleura or the pleural cavity; pericarditis; caries of the ribs or spinal column; mediastinitis; purulent cervical adenitis; suppuration following tracheotomy; diseases below the diaphragm, such as peritonitis, abscess, hydatids of the liver, and retroperitoneal extension of suppurative appendicitis; and infectious diseases. It is frequent in Bright's disease, both when spontaneous and when following scarlet fever. It occurs in connection with acute rheumatism, typhoid fever, measles, small-pox, and congenital syphilis; also in pyæmia and septicæmia, as, for example, when the navel has been the inlet of septic germs, or when a child has been infected before birth with the poison of puerperal fever. A pleural sac filled with serous transudation (hydrothorax) may become inflamed.

The occurrence of bilateral pleurisy, when not due to catarrhal pneumonia, suggests tuberculosis or septicæmia.

The effused liquid is more frequently purulent than sero-fibrinous in childhood (Leichtenstern, Garland),—a fact which Blachez refers to the ravages of scarlet fever.

Pathology and Pathological Anatomy.—The pathological changes do not differ essentially from those seen in adults. The inflamed serous membrane loses its lustre, its blood-vessels become injected, and small ecchymoses appear here and there. The exudation consists of four elements, in varying proportions in each case, but present in all. They are fibrin, serum, leucocytes or pus-cells, and blood.

The predominantly fibrinous exudation is seen in "dry" pleurisy. By it, the pleura becomes roughened and thickened; and it promotes the adhesion of contiguous parts of the pleura to each other. Such adhesions are often found at autopsies in adults, and sometimes in children, where the pleurisy had not attracted attention during life.

The more purely serous or sero-fibrinous exudation deposits a fibrinous coat upon the pleura, of varying thickness in different cases; and it has shreds and flakes of fibrin suspended in its fluid portion. The liquid is transparent, of a light yellow color, sometimes with a greenish tint, and rich in albumen. It often coagulates spontaneously, when withdrawn by aspiration, into a soft jelly. Its specific gravity is 1015–1023, with from four to six per cent. of albumen. The fluid of hydrothorax, on the other hand, is usually below 1015 in specific gravity, and contains about one per cent. of albumen.

From this clear fluid there is every gradation to creamy pus. Indeed, in the same patient, the original sero-fibrinous exudation may become purulent. In cases of pyo-thorax or empyema the tissue of the pleura itself, as well as the false membranes and bands of adhesion and suspended fibrinous coagula, is infiltrated with leucocytes.

Hemorrhagic exudations in children, unless due to trauma, are exceedingly rare. They have, however, been seen in the new-born when infected with puerperal poisoning, and in acute eruptive diseases where there has been a hemorrhagic diathesis.

The amount of fluid which may be exuded varies, of course, with the age of the patient. Lewis Smith states that at the age of four months three ounces of fluid are sufficient to produce complete collapse of the lung. This same amount in a child one year old will give rise to well-marked flatness on percussion. A pint and a half has been found in the left pleural cavity at twenty-two months. Ziemssen found at autopsy two and one-half pounds of pus in the right chest of a girl three years old. Heyfelder removed by thoracentesis the enormous amount of six pints of pus from a boy of six years.

Changes in other thoracic structures may be either mechanical or pathological.

The presence of fluid in the thoracic cavity permits the corresponding lung to retract,—the mediastinum being at the same time drawn towards the opposite side by the other lung. As the amount of fluid increases, actual pressure is exerted. The lung, unless bound down by adhesions, is pressed upward and backward towards the spine of the scapula, where, in extreme cases, it forms a small leathery mass nearly devoid of air and blood.

The heart is pressed towards the healthy side. It may even come to lie in the left axilla, or to the right of the sternum, as the case may be. The heart is therefore interfered with in two ways: first, the compression of the pulmonary blood-vessels on the diseased side impedes the lesser circulation; and, secondly, the heart's cavities and afferent vessels are crowded upon and displaced. Less blood reaches the heart, and to pump that blood through the lungs and into the aorta demands more than the ordinary amount of effort. Hence the pulse in pleurisy with large effusion is small, weak, and rapid, and heart-failure is the chief factor in many fatal cases.

The effusion presses outward, stretching the ribs farther apart and rendering the interspaces plane or convex instead of concave. The circumference of the affected side is increased; but in infants the lungs collapse so readily that little distention of the chest is caused till it is half full of fluid. It should also be remembered that the right side may measure normally a trifle more than the left,—say, half an inch.

The distention also forces the diaphragm downward, and with it the liver on the right and the spleen and stomach on the left side.

The lung on the affected side is superficially inflamed, as was pointed out by Trousseau: so that even in a case of pleurisy we may hear at the upper limit of the effusion the true crepitant râle of pneumonia.

Just as pericarditis may cause pleurisy, so pleurisy may induce pericarditis. If one of these affections be purulent, the other will also be so.

The final result of a sero-fibrinous effusion is that it is absorbed, if the patient lives long enough. If the lung be bound down so firmly by adhesions that it cannot refill its normal position, the mediastinum, diaphragm, and chest-walls are drawn inward, and the spinal column is bent over towards the affected side,—the cavity still remaining being filled by the unexpanded lung and the false membranes, which have become organized. In the course of months and years, much of the deformity disappears; but in the mean time it is obvious that the patient is more than ordinarily liable to bronchiectasis and to tuberculosis.

Purulent effusions practically never are absorbed. Unless operated upon, they cause the death of the patient, or in more favorable instances they discharge, either outward,—empyema necessitatis,—by preference in the second or third space in front, or inward into a bronchus. In the latter case the pus often oozes, as it were, into the lung, in such a manner that air does not escape from the lung into the cavity. Pyo-pneumothorax is very rare in children, unless the air enters through an opening in the chest-wall.

In some few cases the pus of an empyema discharges into the peritoneal cavity; and it may give rise in another way to a purulent peritonitis, by infection carried through the stomata and lymph-channels of the diaphragm, without actual perforation.

Empyema has occasioned a lumbar abscess; it has pointed into the spinal canal, and into the œsophagus.

Symptomatology.—*Pain.*—This is the most prominent of all the earlier subjective symptoms; but it is not so marked in its location, its severity, or its constancy as in the pleurisies of adults, where it is present in at least eighty-five per cent. of all cases. Pain is not infrequently absent in latent pleurisy, and in the varieties affecting feeble cachectic children, and when present is generally most marked in the infra-mammary region of the affected side, but not seldom is found to have a wider range than in adults,—subscapular, subclavicular, axillary, and even umbilical and hypogastric.

It is of extreme importance that this wide distribution of pain, as a subjective symptom in the pleuritic inflammations of children, should be kept in mind, as well as the fact that it is associated with cutaneous hyperæsthesia. These conjoint conditions of pain and of tenderness on pressure over wide areas point to an inflammation of the intercostal nerves or of their neurilemma as a complication of pleuritis in children.

The duration and the intensity as well as the locality of the pain vary greatly in individual cases, at times influencing respiratory movements to such an extent as seriously to embarrass the right side of the heart and to lead to cyanosis, at other times of such an extreme degree of severity as to produce symptoms of collapse, in all cases leading the child to hold his breath, to fix the diaphragm, to arrest the movements of the affected side, and to substitute abdominal for thoracic and costal respiratory movements; at other times pain as a subjective symptom deserves but slight attention by reason of its absence or its presence in merely slight degree.

In severe cases of uncomplicated pleurisy, when the effusion makes rapid progress, pain may disappear on the second or third day; if the effusion is slow in forming, the pain may be equally slow in leaving the child,—six or eight days, or even longer,—and when recurrent it indicates a secondary pleurisy consequent upon a pre-existing tuberculous process or an acute pneumonic complication.

Pain in connection with the frequent purulent pleurisies of children, when accompanied by marked signs of rapid breathing, sudden and increased change in the position of the heart and adjacent abdominal viscera, evidences of greater cardiac disturbances, lividity, cyanosis, cold extremities, feeble and rapid pulse, and impending collapse, warrants the opinion of perforation of the lung, relatively severe in children. Careful examination should be made to determine the presence or absence of pneumo-pyo-thorax in all such cases.

Tubercular and purulent pleurisies are distinguished from sero-fibrinous by the longer duration and the greater intensity of the pain, two conditions which afford a tolerably reliable basis for the diagnosis of such cases.

Fever.—In children the initial rigors, ordinarily slight, usually escape observation. The temperature in sero-fibrinous varieties of the latent type attracts but little attention, and bears but slight relation to the respiration- and pulse-rate, usually varying from 99° to 101° F., and remaining quite constant during the twenty-four hours. Variations from this, in which the temperature continues persistently high, are suggestive of tubercular pleurisies; and when there are marked morning remissions from an evening temperature of 103° or 104° F. the explanation is generally to be found in the change from fibrino-serous to purulent, or in the fact that the pleurisy has been purulent from the earlier or earliest stage of the disease. Still, it must be remembered that the temperature-variations are more marked in children, and the temperature charts of the Children's Hospital in Boston would seem to prove that not infrequently the temperature-curve is of insignificant diagnostic value with reference to the character of the effusion.

Surface thermometry may be resorted to in cases of pleural effusion in children, for the careful experiments of Peter in 1878 proved conclusively the existence of a higher temperature by one or two degrees on the affected side, though above normal on both sides, increasing as the effusion increases, but lessening least on the side involved as the effusion diminishes, the highest temperature having been recorded during the period of greatest pleuritic activity. Peter also noticed the temporary increase following tapping, explained by other observers as due to the pulmonary and pleural congestion set up by the rapid withdrawal of the fluid, a hyperæmia at times quite active, sometimes inducing symptoms of shock, with dyspnœa, an abundant sero-albuminous expectoration, and occasionally — fortunately rarely—symptoms of great gravity, which may lead to a fatal termination.

Pulse.—The pulse-rate bears a more constant relation to the temperature than the respiratory rate does. Influenced largely by the age and the temperament of the child, the pulse in purulent pleurisies may rise to the extreme degree of 160–180 while the respirations are not notably increased. This high pulse-rate, high in the initial stages of the disease, fortunately is most frequently of comparatively short duration,—three or four days,—gradually falling to nearly normal at the end of the first or second week, unless, as is usual in large and rapid effusions, the heart is much compressed or displaced, or both, so that its cavities are but incompletely filled, or great stress is laid upon this organ, in which conditions the pulse remains frequent and feeble. Purulent pleurisies in which the temperature is relatively high induce rapid action of the heart, from the combined influences common to all large pus-formations.

Physical Signs.—The frequent failure in diagnosis with reference to the pleuritic disorders of children is best explained by the difficulty of clearly interpreting the rational signs and by the misleading deductions drawn therefrom. Careful consideration of the evidence presented by physical signs or objective symptoms will make such failures far less frequent, for it has been well stated that in no other disease are physical signs so important for the purposes of diagnosis: this axiom applies with far greater force to the pleurisies of children than it does to similar disorders in adults. From the earliest to the latest stages, through all the periods in the progress or the lapsing of the disease, the physical signs, individually, but with far greater significance when grouped, may be confidently appealed to to establish a diagnosis made doubtful by the perplexing and conflicting elements frequently found to coexist in the rational signs.

Systematic employment of the physical signs for the purposes of prognosis and differential diagnosis involves the careful consideration of all the evidence afforded by inspection, mensuration, palpation, percussion, and auscultation. While recognizing varying values in these diagnostic elements, we are clearly of the opinion that far better results may be reached by following the above-mentioned order, in conducting examinations, than by any other process. To sight, to touch, and to hearing abnormal conditions induced by inflammation of the pleura are clearly revealed, for physical signs often constitute in young children the only means of recognizing this disease.

Inspection.—Pain quickly declares its presence by the expression of the face, the wrinkled brow, the shade of color, depending upon the severity of the pain and its effect, and by the fixation and distention of the alæ nasi, in all cases where the pleuritic pain is so severe as to modify thoracic and costal respiratory movements. Marked deficiency in arterialization produces lividity and cyanosis, while pain, producing shock and collapse, results in pallor. Modification of the respiratory function is proportionate to the amount of pleuritic accumulation and the consequent compression of lung-tissue, and corresponding cardiac pressure and disability. Defective arterialization when present to such an extreme degree as to produce cyanosis must arise from these two causes combined. Inspection of the finger-tips, of the ears and lips, and of the lower extremities reveals the degree of cyanosis, and is of great importance in prognosis and treatment, for whenever present to any marked degree it is a symptom of danger and calls for prompt relief. Sudden pallor is noticed when hemorrhagic exudations have taken place, but a pallor slowly progressing is of no greater significance than that which is produced by other chronic or sub-chronic disorders.

Inspection makes plain the result of the efforts made by the patient to prevent the painful frictional movements of the pleura, in the relative inaction of the affected side and corresponding supplementary respiratory movements of the opposite side: respirations are changed in rhythm and quickened in rate, they are irregular and jerking, and to the largest possible extent are repressed on the affected side. The demand for better oxygenation quickens the rate, which throughout the disease remains notably increased,—in the earlier stages by reason of pain, in the later stages, when the effusion is large, by reason of a deficient aerating surface, owing to retraction and compression of the lungs. Cardiac causes for rapid breathing are also present in large effusions and their effect,—viz., displacement and compression of the heart: when the effusion has reached the high limit of the third rib and second intercostal space, respiratory movements of that side are abolished, inspection showing distention, bulging intercostal spaces, and immobility. The obliteration and bulging of intercostal spaces take place in children far more quickly and from less fluid effusion, but equally in children and in adults it holds true that the retractile energy of the lung, so clearly demonstrated by Garland, is effective in small and midway effusions, arching the diaphragm and permitting the opposite lung to exert abnormal negative pressure on the mediastinum and its contents. When, however, the retractile energy of the lung has been expended through the influence of an effusion large in amount, pressure symptoms and signs arise,—viz., depression of the diaphragm, increased displacement of the heart, and a marked change in the position of the abdominal organs of the affected side.

Inspection of the chest, therefore, shows increased respiratory rate, diminished respiratory excursion of the affected side, modification of rhythm, fixation or distention, or both, of the affected side, with an increase in semicircular and antero-lateral measurements and an increase in circumference of the whole chest. Inspection also shows the influence of effusions on the position of the heart. In the earlier stages and when the effusion is small, the result is always exactly proportionate to the forces disturbing the "equilibrium of traction" by which the heart is maintained in its normal position; in the later stages of large effusions, the position of the heart will be found by inspection to depend upon the degree of hydrostatic pressure exerted, which in the largest effusions is sufficient seriously to impair the power of the heart with reference to pulmonary or systemic circulation. This is aggravated in large left pleural effusions by the partial rotation of the heart upon its base, and the twist given the large venous trunks. Absence of the apex-beat from its normal position, as shown by inspection, becomes therefore, at an early as well as at a late stage in pleural effusions, of great diagnostic significance; and the position of the heart, and the consequent stress laid upon it, may be largely determined by the visible pulsations in abnormal positions.

In children this physical sign is of great importance because of its ready availability, for, in addition to the changes in respiratory conditions brought about by effusions, we find that small left-sided effusions lower the apex towards the epigastrium; as the quantity of liquid increases, the apex describes the arc of a circle whose extremity in large left-sided effusions may be found in the right mammary region, and even in the second and third intercostal spaces of that side. It is in large cardiac displacements, made evident by the cardiac pulsations in the right mammary region, that we also by inspection take note of the combined influences of compression and rotation of the heart and compression of the left lung, in the production of cyanosis, and other signs of defective blood-supply and diminished arterialization,—important guides with reference to operative interference.

Right-sided effusions do not exert so important an influence. The heart's apex is apparently somewhat raised, and at times found in the left margin of the mammary region of the left side, or in extreme cases in the anterior axillary lines.

Palpation.—Ready appreciation of the abnormal variations in lateral as well as in antero-posterior respiratory movements may be had by palpation. We also by this physical sign determine the delay or lagging behind of the affected side, resulting from the repression of the costal movements of the affected side, in the earliest and more painful stages of fibrinous exudation: points in intercostal spaces of greatest pain on pressure may be accepted as localities of greatest pleural inflammation. The largest value of palpation in diseases of the pleura is, however, to be found in the aid it gives, when properly employed, in determining the position of compressed or consolidated lung, as well as the presence of fluid or air displacing the lung, in the modifications of normal vocal fremitus; but, because vocal fremitus depends for its intensity upon the strength as well as the pitch of the voice, great care must be given to the application and interpretation of this physical sign, on account of the high-pitched and feeble voices of children, by which vocal fremitus is greatly lessened and its distinctness impaired: palpation must, therefore, be delicately employed, using the finger-tips only, but with a high appreciation of the pathological fact that the conditions modifying normal vocal vibrations are rarely bilateral in pleural diseases, are usually confined to one side, and generally extend their influence over a large area of chest-wall. Absence of vocal fremitus is conclusive evidence of the absence of lung-tissue, but percussion must be resorted to to determine the precise cause, since pneumothorax also produces the pulmonary displacements which abolish vocal fremitus.

The smaller effusions in children can be determined only by the careful application of all the rational and physical signs, but in the larger effusions palpation supplemented by percussion quickly defines the cause of the abnormal variations. Absence of vocal fremitus is usually caused by fluid; air in the pleural cavity is a less frequent cause. The yielding chest-walls in children, and the intercostal spaces more easily distended than in adults, not infrequently admit of quite distinct fluctuation, when the quantity of fluid is not large, in many places on the anterior and lateral aspects of the chest; in the more localized conditions of surface inflammation associated with purulent pleurisies, fluctuation brought out by palpation is to some extent a guide for local operative measures, although it should be remembered that the point of election in spontaneous openings generally varies from the position chosen for permanent drainage.

Palpation should also be resorted to to confirm the conclusions drawn from inspection as to the position and degree of displacement of the heart, in all cases where the apex-beat is absent from its normal place. Friction-fremitus is not often demonstrable in children.

Percussion.—In the earliest stages of pleurisy, and before there is well-marked evidence of the exudation of fluid and its influence in changing the pitch of the percussion-note, even the gentler forms of percussing elicit from the child expressions of pain, particularly in the vicinity of greatest pleuritic inflammation, and it is not before the effusion mounts two or three finger-breadths that the pitch of the note is raised, and dulness can be made out; with the increase of the effusion there is noted a change to flatness, more slowly than in adults, but distinctly flat in proportion as the fluid increases. When the fluid does not exceed eight to twelve ounces and no adhesions exist, changes of position are followed by changes in lines of dulness; in an upright position the limit of dulness is lower posteriorly and higher laterally and anteriorly than when the patient is in a semi-recumbent position. However, positional changes of level are much less frequent in children, owing to the greater frequency of fibrino-purulent exudations and the consequent formation of adhesions. Percussion does not afford the same degree of accuracy in determining the amount of fluid as in adults, because the vibratory movements of the chest are more easily set up, and the sonority of the lung much more easily brought out, and there is a much readier development of the tympanitic quality of resonance; in fact, it is in the earlier stages to determine the presence of fluid, and in the later stages, when the effusion is large and compression has taken the place of negative pressure or traction, that percussion occupies a prominent position of value as a physical sign; for in the later stages we determine by percussion the outlines of a large effusion and the degree of displacement effected by it of adjacent organs.

Auscultation.—We are able by this physical sign to decide upon the presence or absence of pleural friction-râles, which, however, are far from being the most important of the stethoscopic results, as they are quite inconstant during infancy, and in a large percentage of cases are heard only while the fluid is being absorbed.

Puerile respiration quickly changes to broncho-vesicular and even to bronchial, and from causes relatively slight when compared with those which produce similar results in adults; hence occurs an earlier and more pronounced modification of the respiratory murmur in the earlier stages of pleural effusions in children, and the bronchial quality is also found to be diffused over the greater portion of the chest occupied by the effusion. No explanation of this phenomenon of wide transmission of bronchial respiration is as satisfactory as this, that the pulmonary tissue and the chest-walls of children yield more quickly to the influences developing sonorous vibrations.

Ægophony, by reason of its infrequency, is low in the scale of valuable physical signs. It can be heard only in moderate effusions, and midway between the spine and inferior angle of the scapula, is very inconstant, and disappears in all large effusions when the lung is compressed.

Râles.—The crepitant râle of pleurisy, dependent upon the extension of the inflammation from the surface of the lung to the subjacent pulmonary vesicles, is commonly heard as soon as exudation takes place in the vesicles and bronchioles. This râle may be heard in advance of any satisfactory evidence of pleural effusion, and at times may lead to confusion in the differential diagnosis of pleurisy and lobular pneumonia in their initial stages. Other bronchial râles, when present, are simply those of a coexisting bronchitis, except in the rare cases of pneumonia complicating pleurisy.

Jaccoud classifies the modifications of the respiratory sounds, when the effusion is rising, as follows: diminution of the normal vesicular murmur; no sound other than feeble respirations; broncho-vesicular or bronchial respiration; no sound other than markedly bronchial; cavernous respirations or amphoric, and complete absence of all sound when the lung is compressed and the alveoli are collapsed and the movement of air in the tubes of the affected side is prevented. This series of auscultatory signs reverses itself when the effusion is undergoing absorption.

The voice-sound auscultation-signs are of far less value in children than in adults, and in the pleural diseases of infants and of feeble cachectic children are of little value, because developed with great difficulty even when the pathological conditions are favorable for their transmission.

Constitutional Symptoms.—These symptoms, such as are common to all inflammations, obtain a wide range in children, varying in the severity of their manifestation with the age and general condition of the child and the form of pleuritis present. In feeble and cachectic children, as well as in infants, a frequent type of the disease is the latent or subacute, in which the constitutional as well as the local symptoms are very indefinite and obscure, and, until the disease becomes by the lapse of time chronic, pointing indistinctly to the chest as the seat of the lesion. On the other hand, a child robust and vigorous may be seized with marked indications of profound disturbance of the nervous system: rapid rise in temperature; great restlessness, or the reverse; profound stupor; anorexia and vomiting; rapid and weak action of the heart; rapid respiration: or there may be yet more profound disturbance of the nervous system, as shown by convulsions or symptoms closely resembling collapse.

It is because of this wide range of constitutional signs that the rational signs give so little help in the diagnosis of this disease: the preponderance of reflex influences and results is not infrequently so great as to mask the disease. These are the reasons why so many accomplished clinicians assign but slight diagnostic importance to the subjective symptoms and lay so much stress upon the value of the physical signs.

Diagnosis.—In the earlier stages, and in all circumscribed pleurisies, as well as in those forms in which the exudation is small, the diagnosis is frequently difficult or not made at all. It is in such conditions that the differential diagnosis between pleurisy and pneumonia is of great importance and not easily made out. Pleurisy is more sudden in its onset, is not infrequently preceded by bronchitis, has marked increase in pulse-rate, and respiration though rapid is more shallow and suppressed because more painful; while the temperature-changes are less marked and there is more of local tenderness on palpation than is the case with pneumonia. Due heed as the disease advances should be given to the absence of vocal fremitus in pleuritis with exudation as compared with exaggerated vocal fremitus in pneumonic consolidation, to the sense of increased resistance, greater in pleurisy than in pneumonia, and to the absence of resonance of any degree, or to a high degree of tympany as compared with the percussion-note in pneumonia. Inspection alone may fix the diagnosis, particularly if care be taken to determine the position of the apex-beat, and the influence of pleuritic exudations as tending to displace the heart from its normal position and to disturb the functions of that organ. This is notably true of left-sided effusions; but careful inspection will not fail to develop the results of even moderate right-sided effusions in their influence on the position of the heart.

The differential diagnosis between pleurisy with effusion and hydrothorax rests upon principles familiar to all practitioners. The most important causative influences of hydrothorax will be found in obstructive diseases of the heart and in the diseases which lead to serous transudations. It is extremely doubtful if any rules can be given for the differential diagnosis between serous, sero-fibrinous, and purulent pleurisies. Chronic pleurisy in childhood is more frequently purulent than serous or sero-fibrinous, and although the evidence is very strong that under the age of three years the exudation is in most cases purulent, it is also accepted that aspiration is the only reliable method of differential diagnosis.

Prognosis.—Primary idiopathic pleuritis is seldom fatal. In cases, however, of pleuritis acutissima, where the effusion rapidly fills the chest, and induces powerful compression of the heart, unless prompt relief is given by aspiration, the prognosis becomes quickly grave, from the inability of the heart to perform its work in the systemic and pulmonary channels. Hence a large right-sided effusion may seriously embarrass the thin and yielding walls of the right cavities; or, when the effusion distends the left chest and compresses the left lung, the torsion of the large blood-vessels at the base of the heart, together with the impairment of heart-power dependent upon compression and partial rotation of that organ, may cause death, unless there is a speedy resort to aspiration. The physical signs of grave danger have already been described. The character of the effusion also modifies prognosis, with reference both to immediate and to remote danger, for it is well known that pleurisies in children are much more frequently purulent in their type than in adults.

Secondary pleurisies, or pleurisies which are complicated with other diseases, give rise to a still more unfavorable prognosis: in connection with rheumatism, scarlet fever, or uræmia the pleurisy produces far more marked constitutional disturbance, and influences prognosis proportionately. In purulent pleurisies of long duration the prognosis is grave if there be evidence of the influence of chronic pus-formation in the development of amyloid degenerations of the liver, spleen, or kidney, as shown by albuminuria or increase in size of the spleen and liver. It is also obvious that the prognosis is rendered grave by the long-continued compression of the lung, and by a tuberculous heredity.

Treatment.—The patient should be kept in bed. The room should have a temperature of about 68° F. It should be dry, well ventilated, and on the sunny side of the house.

At the onset of the disease, pain is apt to be severe. Various external applications may be employed for its relief. In a vigorous child, with high fever and dyspnœa, one to three leeches applied over the seat of greatest pain will often be very useful. The number of leeches, and the length of time during which bleeding should be kept up, must depend on the age and strength of the patient and on the effects produced. Two hours' flow will usually suffice. Dry cupping is sometimes very satisfactory.

A poultice of linseed-meal, with one-eighth part mustard, and sprinkled if desired with half a drachm of laudanum, is a suitable remedy for the first, sharp, cutting pains. The continuous use of poultices is, however, not to be recommended. They are heavy; and they require changing every few hours, with consequent disturbance and exposure to changes of temperature. Ziemssen extols cold compresses, of the temperature of faucet-water, covered with some impervious material and changed every five to ten minutes, till the pain abates. He adds, however, that some patients cannot bear them; and they have not attained popularity in this country.

The most satisfactory application, in our opinion, is the following. Four layers of sheet cotton wadding are quilted upon the inside of a merino or flannel undervest split down the front. The innermost, glazed surface of the cotton may be removed. This padded garment is then to be ironed with a hot flat-iron at the bedside, put about the patient, itself covered with oiled silk or muslin, and the whole enveloped in a firm and rather tight cotton swathe. If counter-irritation is desired, the skin may be previously anointed with camphorated oil, either pure or containing one-fourth part of turpentine. This dressing is light and permanent, it supplies a moist warmth, and the mechanical support it gives moderates the painful motion of the diseased side caused by breathing or coughing.

Sometimes an abdominal swathe, by its effect upon the excursions of the diaphragm, gives much relief. It is obvious that mechanical restraint might be carried too far.

The use of blisters is mentioned only for condemnation.

If the pain is extreme and not relieved by the external applications, or if the cough is frequent and troublesome, we must resort to internal remedies. The best is an opiate. The effects of the succedanea of opium are both less satisfactory and less measurable than those of the drug itself. Of course, the younger the child, the greater the caution demanded; but sometimes the narcotic is indispensable. To an infant five to ten drops of paregoric may be given every three or four hours, and to a child of one year thirty minims of paregoric or one-half minim of deodorized tincture of opium at the same intervals. The latter may be dispensed in equal parts of syrup of wild cherry and water. Older children receive a proportionally larger dose. The addition to each dose of a small amount of belladonna is a wise precaution. The following prescription is suitable for a child of four or five years:

℞ Tinct. opii deodorat., ʒss;
Tinct. belladonnæ, ♏v;
Syr. pruni Virgin.,
Aquæ, āā, ℥i.
M.

S.—Shake. Teaspoonful every two hours until relief.

In case the stomach is likely to reject the medicine, a subcutaneous injection of morphine and atropine may be given, $\frac{1}{30}-\frac{1}{20}$ grain of the former and $\frac{1}{750}$ grain of the latter, for a child of five years.

The dangers of aconite are so great and its usefulness is so problematical that we abstain from administering it in the pleurisy of children.

Tincture of ipecac and opium may be substituted for the deodorized tincture recommended above, in case a strong child has a very distressing dry cough.

If the temperature be high,—say, over 103° F.,—a child of five years may receive antipyrin gr. iii–vi, or antifebrin gr. ½–ii, dissolved in water, repeated in three hours if necessary. These may also be given in an enema. A newer drug, phenacetin, has been recommended as a particularly suitable antipyretic for children; but its value and its possible dangerous qualities are yet to be definitely established.

Sponging the entire body, a small surface only being uncovered at a time, with equal parts of alcohol and warm water is also permissible. More than ordinary care should, however, be exercised lest the patient be chilled.

The bowels should not be allowed to remain constipated, because distention of the abdomen aggravates the patient's dyspnœa.

In the beginning of the disease the digestive powers must not be overtaxed. Milk is the best diet, diluted if necessary with lime-water or plain water. Gruel is also suitable. It is not wise to curtail the amount of liquid ingesta, with the object of preventing or diminishing an effusion, in children.

In the second stage, when an effusion has already taken place, the diet should be sustaining and nutritious. Meat broths, beef juice, soft-boiled eggs, milk toast, and the numerous farinaceous puddings may now be added to the previous list; and a moderate amount of sherry, port, or other alcoholic stimulant should be given, if indicated by the pulse. The bitter tonics, such as elixir of calisaya, or compound tincture of cinchona or of gentian, with syrup of orange-peel, may be employed to stimulate appetite and digestion. If there is anæmia, the tartrate of iron and potassium, dissolved in water, with one-eighth part of glycerin to prevent its decomposition, is suitable; or the citrate of iron and quinine; or the syrup of the iodide of iron, of which five to twenty drops, largely diluted, should be given after meals, through a glass tube.

Eustace Smith believes that iodide of potassium has been efficient in his hands in promoting absorption. He gives from five to eight or even ten grains every six hours to a child of four years; and at the same time he employs counter-irritants. Tincture of iodine, or the stronger linimentum iodi, B. P., is painted every night, after the fever has abated, upon a small area, until that spot begins to be inflamed, when a new one is chosen. Another external application at this stage is unguentum iodi, diluted with an equal part of lard and rubbed over the affected side morning and night. We doubt the efficacy of any of these external aids to absorption.

If the pulse becomes at any time feeble and rapid, digitalis is invaluable, ʒss–i thrice daily of the infusion, or of the tincture ♏iii–v, at the age of five. A child two years old can take one minim of the tincture every three hours. If the urine be scanty, we may combine with the above acetate or citrate of potassium, in the dose of five to ten grains, dissolved in syrup of lemon. Another prescription is,—

℞ Spiritus ætheris nitrosi, ʒii;
Liquoris potassii citratis ad ℥iii.
M.
S.—Two teaspoonfuls in water every two hours for a child of five.

If the skin be very dry, the effect of giving the patient a warm full bath for fifteen to thirty minutes, followed by wrapping him warmly in heated blankets, may be tried, in the hope that the diaphoresis may hasten absorption. This procedure should not, however, be repeated often, for fear of debilitating the child. Drastic purgatives are to be discarded, for the same reason.

Thoracentesis.—If the effusion remain for more than two weeks undiminished, or if at any time there is cyanosis or orthopnœa, or if the effusion fills the chest up to the second rib, thoracentesis is indicated. Unusual circumstances which would hasten such interference are a bilateral effusion, or a complication with pericarditis, heart-disease, pneumonia, or severe bronchitis. The danger of delay when the operation is indicated is much greater than the danger incurred by doing it prematurely; but it should be remembered, on the other hand, that in children particularly absorption is often speedy when it once begins, so that the least sign of improvement should be valued. As the appetite and the fever are generally improved at almost the same time with the commencement of absorption, their condition may help us in determining upon interference or expectancy, as the case may be.

If the behavior of the temperature, the severity of the general symptoms, or œdema of the chest-wall lead us to suspect empyema, there should be still less delay than otherwise would be the case. Here, or where the differential diagnosis is not absolutely established,—*e.g.*, between liquid effusion and solidified lung,—a convenient and safe resort is the subcutaneous syringe. If pus be found, there is but one thing to do,—that is, to remove it. If the pus be laudable, aspiration should be tried, and repeated once or twice; but if then the empyema persists, a permanent opening is demanded. If the pus be fetid, aspiration should be abandoned at once in favor of the knife.

The Apparatus for Thoracentesis.—Without enumerating all the devices for performing the operation, we will briefly describe the arrangement which seems to us the best. The receptacle into which the fluid is to be drawn must be provided in some way with means to create within it a vacuum previous to the puncture. The rubber tube through which the fluid is to flow into this receiver should be connected with it by a stopcock. Near the opposite end of the rubber tube it is convenient to have interpolated a bit of glass tubing, so as to see the escaping fluid at once as it flows out. With regard to the piercing instrument, a sharp, hollow needle is objectionable, because it may prick the lung as the latter expands. The needle should therefore have some contrivance for guarding its point after entrance. Better still is a trocar and canula with its outward extremity supplied with two cocks,—one at the side, to which the outflow-tube is attached, and one at the end, through which the trocar is withdrawn; whereupon it is closed. It is very advantageous to have a probe sliding in an air-tight joint, to fasten upon the end when the trocar is removed, so that if coagula obstruct the canula the cock may be opened and the probe used to clear the canula and yet no air enter. We prefer a medium-sized trocar to the very small ones recommended by many. They cause but little more pain, and are less apt to be broken or plugged up. The rapidity of the outflow can be regulated by the cock at the entrance to the receiver.

The Operation of Thoracentesis.—It need hardly be premised that the hands of the operator, the skin of the diseased side, and the instrument employed should be surgically clean. The little patient should be supported in a sitting posture by the nurse. Ether is unadvisable; and the discomfort and apprehension caused by chilling the surface at the intended point of puncture more than counterbalance the slight relief thus afforded. The point of election for aspiration is an inch or two outside the angle of the scapula, in the seventh or eighth space. Placing the left thumb-nail just below the selected spot, the needle should be gently but quickly pushed across its edge and through the chest-wall, close to the upper border of the rib. The artery, it will be remembered, runs along the lower border. The quickness of the thrust enables the instrument to pierce, rather than to push before it, any false membranes which may exist. A necessary caution is, not to drive the trocar too deeply: one inch is the ordinary limit. The fluid should not be withdrawn rapidly; and the operation should cease if there is coughing, pain, or dyspnœa. The removal of a few ounces or even drachms is sometimes followed by the rapid absorption of a large effusion previously stationary.

After the operation, a little stimulant may be required, or an opiate to check the cough, if distressing.

Convalescence.—The patient should be got out of doors as soon as possible, and every means should be taken to invigorate the system. A change of climate is often useful. There should be no undue haste about a return to school. If the lung has not expanded completely, exercises or plays calculated to develop the chest should be encouraged, preferably, of course, in the open air,—*e.g.*, mountain-climbing. Several times a day the patient should inspire forcibly, at the same time restraining the healthy side as much as possible. Massage of the chest-walls is also recommended.

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EMPYEMA.

By A. T. CABOT, M.D.

Suppurative pleuritis is a rather common disease of childhood, and, according to some statistics,[1] is more prevalent in the first five years of life than at any subsequent time. The tendency to the disease apparently reaches its height at about the fourth year, and after that gradually diminishes with increasing age.

Pleuritic effusions are far more apt to become purulent in children than in adults. In fact, very nearly one-third of all effusions into the chest in childhood may be expected to consist of pus or sero-pus.

Although purulent effusions may appear spontaneously in the chest, without any discoverable exciting cause, yet in the majority of cases they are secondary to some other disease, either general or local.

Pneumonia stands in the front rank as a predisposing cause for empyema: even the cases occurring in the course of a typhoid fever or a rubeola are usually preceded by an inflammatory process in the lung. Scarlatina may be mentioned next, and then tuberculosis. In the latter case the tuberculous process may be general or may at first be confined to the pleura.

Among the less frequent causes are diphtheria, whooping-cough, and traumatic occasions, including caries of the ribs or vertebræ.

Purulent effusions into the chest also occur as a consequence of septic absorption, either with or without inflammation of other serous membranes. Heubner[2] has described a form of the disease occurring in infants, in which several serous surfaces are affected with a purulent inflammation, and the disease, by its sudden onset and rapidly fatal course, as well as by the discovery of abundant colonies of micro-organisms in the lymphatics, lungs, and kidneys, is shown to be of a zymotic character. In the absence of any wound through which absorption could take place, the milk has been suspected as the vehicle of contagion in these cases.

The effusion is usually a general one. The absence of previous attacks of inflammation in the chest makes the occurrence of encysted empyema rare in infants. In older children, who have suffered from previous pleurisies, the cavity may be divided by adhesions and the fluid may thus be encysted.

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[1] Bouveret, Traité de l'Empyème, Paris, 1888.

[2] Jahrb. für Kinderheilkunde, 1884, Bd. xxi.

An inflammation of the pleura may be purulent from the start, but far more commonly it begins with a serous or a sero-purulent fluid, and this changes more or less rapidly or insidiously into pus. There is not infrequently a considerable quantity of clotted fibrin in the pleura in these cases.

If the pus exists for a long time in the chest, it may, by its pressure, cause caries of the ribs. More commonly, however, it breaks down the soft parts and either finds its way to the surface through an intercostal space (empyema necessitatis) or perforates the lung and discharges through a bronchus. Occasionally it passes down through the diaphragm, and either, discharging into the abdomen, sets up a peritonitis, or, if cut off from the general peritoneal cavity by adhesions, finds its way somewhere to the surface.

When an empyema has existed for some time, the pleura becomes much thickened, and of hard, fibrous consistency. If the lung has been long compressed, it may be changed into a firm, carnified cake, pressed back against the upper and posterior wall of the chest, and without a vestige of alveolar structure.

Subpleural inflammation may associate itself with the rapid, acute empyemas of childhood, and we sometimes see subpleural abscesses under these circumstances.

It is not intended here to go at length into the symptomatology of empyema, as the symptoms, so far as they depend on the inflammation of the pleura and the presence of fluid in the chest, have been sufficiently considered under the head of Pleurisy.

It may be worth while, however, to call attention to the occasional insidious character of the disease, especially in very young children, and to emphasize the importance of always examining the chest of an infant, even if evidence of pulmonary disease is wanting or consists only in a slight dyspnœa. The frequent occurrence of empyema as a complication in the course of acute diseases is especially to be borne in mind in this connection; and frequent physical examinations alone will save from the error of overlooking it.

If the chest is found to contain fluid, its purulent character may be suspected if it has accumulated with great rapidity and if it is accompanied by high fever and great prostration. The change from a serous to a purulent fluid may be marked by a sudden aggravation of symptoms and an increase or return of fever. On the other hand, if an effusion has existed for a long time in the chest of a child, even without fever, the chances of its being purulent are very great.

Fortunately, we have in the exploring-needle a safe and sure means of deciding upon the presence and character of the fluid. In every effusion which does not speedily disappear, aspiration should be used.

While it is true that children recover after operations for empyema more rapidly and more completely than is the rule in adults, still, in cases not operated upon the disease is more rapidly fatal in childhood.

Leichtenstern[1] considers the escape of the pus, either by operation or by a spontaneous opening, as a *conditio sine qua non* of recovery. He thinks that perforation into the bronchi is a common occurrence, often overlooked, and that it explains many of the cases of apparent spontaneous resorption of the pus. He points out the fact that the opening into the lung often appears just after an aspiration, and thinks that the expansion of the lung opens a commencing perforation.

While this course of things is, no doubt, true in many cases in which it is overlooked, still, there are others in which there can be little doubt that pus remains in the chest, becomes slowly absorbed, and leaves a cheesy residue such as is found in connection with other abscesses.

As we can never properly count upon a spontaneous cure or a recovery following internal medication alone, we will proceed at once to the consideration of the various methods advocated by good authorities for the removal of the pus.

In the treatment of empyema in childhood we have a somewhat different problem from that which presents itself in adult cases. This is owing to the greater curability of the disease in childhood. Adults rarely recover, in fact may be said never to recover, without the establishment of a free opening, either spontaneously or by operation. Children, on the other hand, are reported to recover occasionally by spontaneous absorption of the pus; and it is not a very uncommon experience with them to see a large residue of pus, left after one or two aspirations, slowly disappear by absorption. In such cases a diminishing area of dulness gives evidence of the shrinking effusion, and it may be years before the restoration of respiration in the base of the lung is complete. Occasionally, in exceptional cases, the absorption of the fluid is much more rapid and the dulness soon completely disappears.

That all of these cases are to be explained by the supposition that the pus is discharging itself through the lung, as Leichtenstern affirms, may well be doubted, but this course of events should always be suspected and watched for.

The occasional successes of treatment by aspiration encourage many practitioners to treat their cases in this way and so avoid the more radical measure of a free incision. It is almost impossible to determine what the percentage of cures from aspiration is. Successful cases are reported, while the unsuccessful ones are often not recorded. A large proportion of the failures of aspiration are finally treated by incision, and a want of success is sometimes scored against pleurotomy which is due rather to the delay and consequent loss of strength while aspiration was being tried.

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[1] Gerhardt's Handbuch der Kinderkrankheiten.

Furthermore, the final history of the cases of recovery is important in deciding upon the value of the operation. If the cheesy residue of the pus subsequently sets up a general tuberculosis, or a local inflammatory process in the lung, the incompleteness of the operation is directly responsible. Or, if the long convalescence while absorption is going on leaves a puny and weakly child, this result should not be counted a success when compared with the quick and complete recovery following a free incision.

While, therefore, it is conceded that aspiration will sometimes cure empyema in children, we should consider, in any given case, that in using this method we are subjecting our patient to a delay which may be serious; that we are giving the lung time to contract adhesions which may prevent its full expansion; and that, finally, the best result that we can hope to obtain is an imperfect one, which may leave the nidus for a future tuberculosis.

Bearing these facts in mind, we should not long persist with the needle unless the effusion shows a distinct tendency towards absorption. This will not often be found to be the case except when the fluid is sero-purulent and approaches closely to a simple pleurisy.

If it has been decided in a given case to try to bring about a cure by aspiration, it is necessary to observe certain precautions.

The needle may be introduced through a portion of the skin which has been rendered insensible by the injection of a cocaine solution or by the action of cold. With young children it is difficult, even with good local anæsthesia, to keep them quiet, and it will often be necessary to give ether. In such a case, if several aspirations are found to be necessary, it will be better to make a free incision and so spare the child the depressing effects of repeated etherizations.

In selecting a needle, it should be remembered that the pus may be thick and contain many little clots: a needle of fairly good calibre should therefore be chosen. It is usually not well to puncture at the most dependent part of the chest, as here the clots settle and will be likely to clog the tube. Moreover, it is to be remembered that in children the diaphragm rises higher than in adults, and that especially on the right side the liver is to be looked out for. A good point to choose for the puncture is the posterior axillary line in the sixth interspace on the left side, and the fifth or even the fourth interspace on the right.

The point for the puncture having been selected, the arm should be drawn firmly upward, to make the separation of the ribs as great as possible, and, with the left forefinger pushed firmly into the interspace as a guide, the needle is quickly thrust in alongside of it. If care is not taken in this way, a slight movement of the chest-wall may so shift the relations that the needle will strike one of the ribs instead of the space between them, thereby causing much additional pain.

The pus may be drawn out by an aspirator into a Potain's bottle; or, if a rubber tube is attached to the needle and the end is dropped into an antiseptic solution on the floor, the weight of the column of fluid in the tube will empty the chest by a gentle and gradual suction.

If troublesome cough or dyspnœa supervene, the aspiration must be stopped; but an effort should be made to remove the pus more thoroughly than is necessary with a serous fluid. This end may best be attained by drawing it out very slowly, so as to expand the lung gradually.

Accidents from heart-failure during aspiration are much less common in children than in adults.

Statistics collected by Bouveret show that it is in very young children, and in cases of recent origin, that success may be looked for from aspiration. If a cure does not follow two or three aspirations, the attempt had better be abandoned in favor of more radical measures. To continue aspirations, as some advocates of that system urge, to twenty or thirty (in one case to one hundred and twenty-two times) seems a triumph of endurance on the part of the patient, and of perseverance rather than of good judgment on the part of the physician.

In aspiration, as in every other operation, strict antiseptic precautions should be observed. A neglect of them in the first instance may cause a serous to change to a purulent effusion.

Surgeons have sometimes modified the method of repeated aspiration, by injecting antiseptic solutions through the needle and again withdrawing them, thus washing out the chest. Tincture of iodine has also been thus used as an injection. The incompleteness of this method of irrigation, the impossibility of completely withdrawing the fluid injected, and the extreme susceptibility of children to the poisonous effects of antiseptics, especially of carbolic acid, make this use of injections a measure of doubtful utility and sometimes of positive harm.

On the intermediate ground between aspiration and the free incision we have several procedures attempting a sort of continual aspiration, of which that known as Playfair's method may be taken as a fair example.

A drainage-tube is introduced into the chest through a canula thrust between the ribs, and when the canula is withdrawn the tube is held in place by the soft parts clinging about it. This tube is conducted into a bottle by the bedside, where it dips beneath an antiseptic solution. The siphon action of the tube constantly draws the pus out of the chest.

The disadvantage of this contrivance lies in the fact that the tube soon becomes loose in the soft parts, so that air can enter the chest and put a stop to the siphon action. It is almost impossible, also, to conduct this treatment aseptically. These difficulties, together with the frequent stoppage of the tube with clots, form serious objections to the method; and, although cases sometimes get well under this treatment, it is very unreliable, and is about as severe an operative procedure as the open incision.

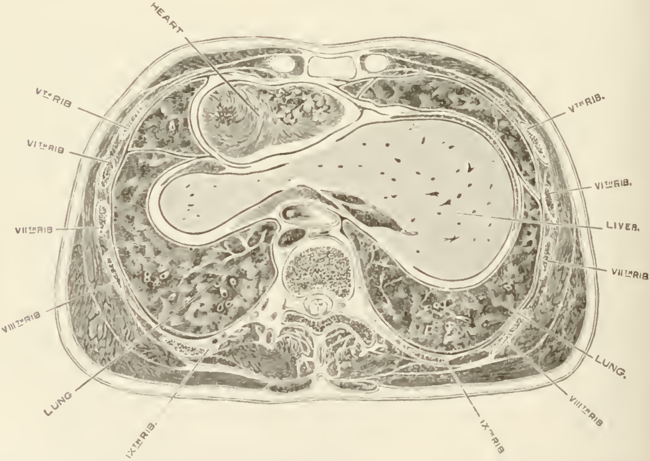
The antiseptic method has robbed pleurotomy of most of its old dangers, and with proper precautions a free incision into a chest full of pus is followed by the happiest results.

To the question, What are the proper precautions? it is hard to give a settled answer in the matter of some of the details.

The necessity for absolute surgical cleanliness of the operator, of the patient, and of the instruments or hands that are to come in contact with the patient, is generally acknowledged. In regard to the value of the antiseptic spray in this operation, and the importance of disinfecting the air which is sucked into the chest, there may be difference of opinion.

The writer showed, some years ago, that by placing an efficient steam spray-producer near the patient the air about the chest might be so displaced that only an antiseptic vapor should enter the opening. The operations done in this way were extremely satisfactory in the speed of their recovery, and theoretically this protection seems of decided value. More recently a series of cases operated on without the spray have shown that quick recovery may also be obtained without the added protection of carbolized vapor. Nevertheless, in this last series there were certain cases which it was felt would probably have done better under the more complete protection of the spray, which still seems of a certain degree of advantage in promoting quick cures. Certainly, in operating without the spray one rarely sees the discharge change almost at once to serum, as was not uncommon after operations done with it.

FIG. 1.



This figure (after Braune) represents a frozen section of the body at the level where an opening into the chest for drainage is usually made. It shows the encroachment of the liver on the chest-cavity at this level, and the consequent importance of care, in making a puncture or incision, to avoid injury of the diaphragm, or even of the abdominal organs. The difference in the relations of the liver to the right and left chest-cavities is also worthy of note.

Its great disadvantage lies in the chilling effect of a strong current of vapor thrown against so large a surface of the body as is exposed in this operation. This so adds to the shock of the procedure that it should be avoided in feeble patients, and even in those who are reasonably strong it should be diminished as much as possible by protecting the body from this cold blast.

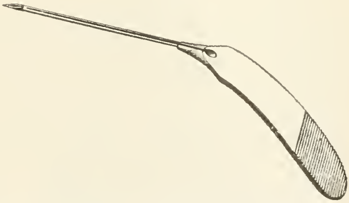
To promote the most perfect drainage, it is obvious that the opening should be made in a dependent part of the chest, and, further, in a part that shall be dependent both when the patient is upright and when he is horizontal.

These requirements are best met by an opening in a vertical line below the posterior fold of the axilla and between the sixth and seventh or the seventh and eighth ribs. It should be remembered that in children the diaphragm rises higher into the chest than in adults, and therefore, unless there is good evidence that the effusion has pressed it below the normal position, the opening will be more safely made in the sixth than in the seventh interspace.

In case of any doubt, it is well to establish the presence of pus at the point chosen, by a preliminary puncture with an aspirator or exploring-needle, before making the incision.

The writer has found a needle like that in Fig. 2 a convenience for this exploration. It is hollow, with one side cut out, and has the advantage over an ordinary grooved needle that the elastic tissues do not press into the groove and prevent the escape of pus along it. If pus appears, a probe-pointed bistoury can be slipped along the groove, and with it a sufficient opening can at once be made. The chest is thus opened thoroughly at one cut, without dissection. This speed of operating is a great advantage in feeble subjects or in cases where local anæsthesia is used.

Fig. 2.



Grooved needle for exploration, which serves as a guide for the knife when pus is reached.

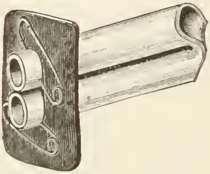
The incision is to be made as near the middle of the intercostal space as possible, and should be carried along parallel to the ribs: in this way injury to the intercostal arteries may be avoided.

To insure thorough and constant drainage, two tubes should be inserted, and they should be of as large a size as the intercostal space will admit. It rarely happens that the ribs are too close together to allow of the introduction of the tubes. If this is the case, however, a segment of a rib may be removed, or a rib may be perforated with a trephine.

The tubes should not project far into the cavity of the chest, but should barely reach through the wall. They may be held in place by safety-pins that rest on a rubber shield in the manner shown in Fig. 3, the whole being fixed by a strap of adhesive plaster.

If there is much fetor, or if the chest contains large clots, the cavity should be syringed out. Considerable care in thoroughly removing the masses of fibrin is amply repaid by the subsequent quick recovery.

FIG. 3.



A double tube made by cutting an opening in one side of the tubing and bending it at that point. A rubber shield is then slipped on over the ends of the tube, which are held by safety-pins resting on the shield. The whole is held in place by a swathe of adhesive plaster.

For injection into the chest it is important to select a solution which is innocuous if partly retained. Carbolic acid is highly poisonous under these circumstances, even in very dilute solutions, and should never be used. Corrosive sublimate in a strength of one to eight thousand or ten thousand is less to be feared than carbolic acid, but if retained in considerable quantity might lead to a dangerous degree of absorption. Phenyl (sulpho-naphthol), in the proportion of one part to fifty or one hundred parts of water, may be used. Perhaps, however, the best solution for this antiseptic irrigation is made by adding one part of liquor sodæ chlorinatæ (Labarraque's solution) to fifteen or twenty parts of water. This is a powerful antiseptic and deodorizer, has no poisonous properties, and is not an irritant. If used in too strong a solution, it sometimes forms soft coagula with the serum and may clog the tubes; but in the strength mentioned this does not occur.

When the cavity is pretty well emptied, the dressing may be applied. In considering the application of this, it may be well to review briefly the mechanical principles involved in the expansion of a lung that has been compressed by fluid in the chest, and to see how this expansion may be favored by an appropriate dressing.

Suppose a case of empyema in which an opening has been made and the pus allowed to escape. Upon the removal of the pressure the lung at once expands somewhat by virtue of its own resiliency and by the partial re-establishment of its circulation. Further, each contraction of the chest with closed glottis (cough or sneeze) presses the air from the well side over into the affected lung, partially expands this, and so forces the air or fluid in the pleural cavity out through the opening in the side. When the cough is over, and the chest again expands with a forced inspiration, air rushes back to take the place of that just expelled. There are two avenues by which this returning air enters the chest,—namely, the bronchus of the lung and the opening into the pleural cavity.

If this latter opening be as free and unobstructed as the bronchus, the air has as ready access to the pleural cavity as to the bronchial tubes, and, the pressure on the outside and inside of the lung being thus equalized, it resumes its condition of semi-collapse.

If, however, the opening in the side is narrowed by the obstruction of a dressing, or, later, by the closing in of granulations, the air returning after a forced expulsion is somewhat opposed in its entry into the pleural cavity, while the bronchus admits it freely, so that the atmospheric pressure inside the lung is somewhat greater than upon its outer surface, and the dilatation effected by the cough is more or less maintained. It is thus that the first expansion of the lung takes place under the usual dressings of oakum or other absorbent material.

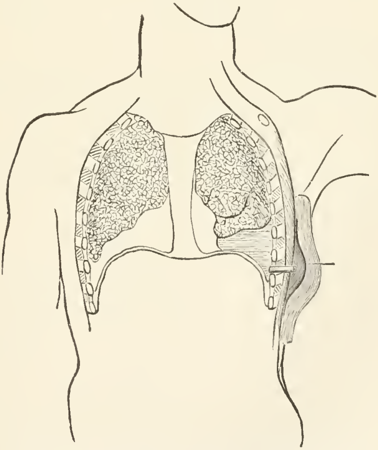
This dilatation of the lung is likely to be interfered with by a provision of nature which here may act detrimentally to the healing process. I refer to the adhesion of the inflamed pleural surfaces when brought in contact.

Of course, if the surface of a lung only partly dilated becomes firmly fixed to the parietal pleura, its further expansion is greatly interfered with, and may become impossible. It is, therefore, very important to induce the lung to dilate to its fullest extent as soon as possible, so that the pleural adhesion, when it occurs, may bind things in their proper positions. This rapid dilatation may be powerfully assisted by a proper dressing.

The problem is, to provide for the easy escape of air and fluids from the chest, and to obstruct the re-entry of air into it. A Lister dressing, rightly applied, fulfils the required conditions thoroughly. The proper method of its application is as follows.

The tubes being secured in place and cut off so that their ends project just above the wall of the chest, a handful of loose gauze, wrung out in an antiseptic solution, is placed around and over them, and over this a piece of mackintosh large enough to project in every direction beyond the gauze beneath it. Over this, again, are placed many (twelve to fifteen) layers of dry gauze, and lastly a sheet of cotton batting to provide for equal pressure. (Fig. 4.) This whole dressing is held in place by a gauze or flannel bandage, some of the turns of which should go over the shoulder, to prevent its slipping down.

Fig. 4.



This diagram shows the proper arrangement of the dressing. The dark line A represents the cross-section of the mackintosh.

The pump-like action of this dressing is due to the method in which the mackintosh is applied. This rubber layer, impervious to the air, overlaps the gauze beneath it so that its edges are held closely applied to the skin by the elastic pressure outside. How closely it clings to the skin can be appreciated only by one who has often removed these dressings.

When, now, air is forcibly driven out of the chest by a cough or other effort, it lifts the edge of the rubber and escapes, but, as the elastic outside dressings immediately press the mackintosh again to the side, the air which could lift it from within cannot get beneath it from without. It acts, in short, as a valve, and, with the aid of the movements of the chest, pumps the fluids and air from the pleural cavity.

If the suction is working efficiently, it ought to be found at each change of dressing that all of the pus is in the gauze and that practically none is retained in the pleural cavity. When this is not the case, and a considerable residue of pus is retained in the chest, it is due either to the fact that the lung is so tied up that it cannot expand and force out the pus, or to some obstruction to the flow through the tube. In the latter case, if the chest is free from clots, and the air passes freely in and out during the time the dressing is off, the obstruction is probably due to the pressure of the dressing over the ends of the tubes; and this condition should be carefully guarded against by surrounding the ends of the tubes with a thick ring of gauze.

Under this form of dressing, if the lung is not already so tied up that it cannot expand, we may look for its rapid dilatation and the quick closure of the cavity. The discharge soon becomes serous, and in the course of a week or ten days is reduced to a drachm or two in the twenty-four hours. One of the tubes may now be removed, and when the discharge dwindles to a few drops a day—when, in short, it is no more than would be expected to come from the sinus through the chest-wall—the last tube can be left out.

It is important to get the tubes out as early as can safely be done, as there is danger that they will establish a chronic sinus, or that by their long pressure they may set up caries of a rib.

When the lung does not fully expand to fill the chest, in consequence either of its being disabled by long compression or through its becoming adherent in a faulty position, there is left a cavity of greater or less size to be closed by some other process.

This cavity, like that of any abscess, is surrounded by a wall of granulations; and, as this gradually changes into fibrous tissue and contracts, it closes down upon and diminishes the size of the abscess, and at the same time draws in and displaces the neighboring organs and parts. The mediastinum and the heart with its great vessels are pulled over, the abdominal organs and the diaphragm are drawn up, and the chest-wall falls in as far as the stiff framework of the ribs will allow.

Fortunately, in children the tissues are soft and pliable, the ribs are not so unyielding as in adults, and healing by this gradual contraction of the cavity is therefore much easier and more likely to occur than later in life.

When, however, in late childhood, the walls of the chest do not sufficiently yield, and a cavity remains, the surgeon may be compelled to resort to resection of the ribs to allow of a sufficient falling-in of the chest to bring the walls of the abscess together.

This operation is best performed on the side of the chest. The ribs are here more accessible than either in front or behind, and considerable segments can be removed without seriously interfering with important muscles.

Various incisions have been used for uncovering the ribs to be operated upon. Estländer makes his cut over the middle of the intercostal space and parallel with the ribs. Through each such incision the ribs above and below it are resected. Thus, for the removal of portions of six ribs, three separate incisions would be required. Other operators have preferred large curved cuts forming flaps, and Trélat has used an I-shaped incision.

Owing to insufficient nourishment of the skin, flap operations on the sides of the chest are liable to be followed by more or less sloughing.

If the cavity is not very large, a straight incision at right angles to the ribs will often afford sufficient room. The skin of the chest is so easily pushed from side to side that portions of rib from four to seven centimetres in length can be removed through a vertical cut. If at the lower end a sufficiently long piece of rib cannot be uncovered, a cross-incision may be added.

When the ribs are exposed, the periosteum is slit up over the portion to be removed, and with a blunt-pointed, somewhat curved elevator it may be readily stripped back, so that the section is done subperiosteally and much hemorrhage is avoided. The rib is then cut out with bone-forceps.

When the chest is already much contracted, it may be quite difficult to free and separate the first rib attacked, and sometimes the saw or chisel is required for its section. After it is removed, however, the others are easy of access.

In deciding upon the proper lengths of the pieces of rib to be removed. the depth and shape of the cavity must be considered. This can be quite accurately determined by exploration with probes or with the finger after the operation has enlarged the opening.

Most operators agree that portions of all the ribs lying over the cavity should be removed. This is a rule not without its exceptions. It is, however, true that there is more danger of want of success from doing too little than from doing too much.

After the chest is widely opened by the resection of the ribs, the lining wall of the abscess may be curetted with a Volkmann's spoon, thus removing the fungous granulations, false membranes, and cheesy clots. This often assists greatly in promoting rapid healing. In case of a small abscess, the cavity may be filled with tampons of iodoform or other antiseptic gauze, and very good results are reported following this proceeding.

Finally, before the wound is closed, thorough drainage must be provided. If the first opening is not in the most dependent part of the cavity, a second should be made there, and thus drainage established.

PERIPLEURITIC ABSCESS.

Abscesses in the thin layer of cellular tissue between the parietal pleura and the chest-wall are of rare occurrence. They may arise primarily without apparent exciting cause, but are more commonly secondary to empyema or follow fracture or caries of the ribs.

An abscess following an empyema may communicate by fistulous tracks with the pleural cavity. When, however, the suppuration starts in the peripleural tissue, it does not tend to break through the pleura, but does have a tendency to open outward, between the ribs, sometimes by several fistulous openings.

The diagnosis between such an abscess and a small empyema-cavity may be very difficult, especially when the trouble is located in the lower part of the chest. Encapsulated collections of pus, when bounded below as well as above by lung-resonance, should always lead to the suspicion of a peripleural abscess.

Treatment should consist in the early and thorough evacuation of the pus, with drainage of the cavity.

FRACTURES OF THE CHEST-WALL.

Owing to the great elasticity of the bones and cartilages, fractures of the sternum and ribs in childhood are of very rare occurrence. These injuries are almost always the result of great crushing violence, as when a wheel passes over the body or when a child falls from a height.

If the sternum is thus injured, the separation occurs, as a rule, between the segments of the bone, which are not ossified together until after puberty; and the lesion is a diastasis rather than a fracture.

The ribs may be broken with a perceptible separation, or may suffer a green-stick fracture with an absence of deformity. The separation of a rib from its cartilage is also occasionally met with.

When there is a distinct separation between the fragments, the diagnosis of these injuries is easy; but when, as is often the case, there is no deformity, the fracture or diastasis is to be inferred from the persistence of pain and tenderness at the point of injury. Crepitus between the ends of the bones may be perceptible to the hand or to the stethoscope during the respiratory motions of the chest. Emphysema or hæmoptysis occasionally also gives evidence of injury to the lung by sharp fragments; but these complications are far less common in children than in adults.

The treatment of these injuries should consist in the immobilization of the chest-wall with a swathe of adhesive plaster.

In diastasis of the sternum it may be difficult to reduce the deformity and afterwards to keep it in place. There is often considerable overlapping of the bones. The patient should be laid on the back on a flat bed, with a cushion under the chest, so as to put the trunk in a position of extreme extension. A sudden cough or sneeze may accomplish reduction when the body is in this position. A pad over the point of injury, with a swathe of adhesive plaster, will help to keep the bones in place after they are reduced.

In compound fractures of the chest, in addition to the measures for keeping the bones in place, careful antiseptic precautions should be observed in the treatment of the wound. If we can prevent suppuration, we avoid the dangers and trouble incident to the accumulation of pus in the chest.

When the fracture involves the sternum, a close watch should be kept, in order that the formation of pus in the mediastinum, if it occurs, may be early detected. If it does form there, it must be freely evacuated, even by trephining through the bone when necessary.

CARIES OF THE STERNUM AND OF THE RIBS.

Caries of the sternum is rare in children. It may follow a fracture, but more commonly it appears as a local manifestation of tuberculosis or congenital syphilis. As it occurs usually in unhealthy subjects, it often proves obstinate in its resistance to treatment; and if it leads to suppuration in the mediastinum it may seriously threaten life.

The strength of these patients should be supported in all possible ways, by judicious feeding, by tonics and cod-liver oil, or even, when feasible, by change of air. All abscesses and sinuses should be freely opened and curetted; the carious bone must be thoroughly removed, with a sharp spoon or burr drill; if the mediastinum contains pus, it should be opened with a trephine at as dependent a point as possible, and thus carefully drained.

Caries of the ribs is more common than that of the sternum. It, too, follows injuries, or appears as a consequence of tuberculosis or syphilis. The pressure of the pus in empyema sometimes leads to erosion of the ribs, and the prolonged presence of a tube in an intercostal space may lead to a limited caries about it.

Treatment consists in the thorough removal of the diseased bone and in provision for the free escape of the pus. Resection of the rib may be practised in case of extensive disease; when the caries is of limited area it may be removed with a curette.

TUMORS OF THE CHEST.

Tumors of the lung, even when they start upon the pleural surface, are so difficult of detection that they do not offer opportunities for surgical interference. When, however, a tumor growing from the chest-wall extends to the lung, this circumstance does not necessarily prevent its removal. Portions of the lung may be excised without causing serious hemorrhage.

Unfortunately, the tumors which thus extend to the lung are of a malignant character, and almost inevitably return, either locally or in some distant part of the body: so that there is not much encouragement to the surgeon to undertake the formidable task of their removal.

The tumors of the chest-wall which may demand surgical treatment take their origin either in the bones, in the periosteum, or in the cartilages of the ribs. Those which thus occur in childhood are usually sarcomatous in character, although bony, cartilaginous, or fibrous growths may arise.

If any of the malignant new growths are thoroughly removed, their recurrence need not be expected.

Should a sarcoma of a rib, either starting centrally in the bone or growing from the periosteum, be discovered before it has outgrown the possibility of thorough removal, its extirpation should be attempted, and a considerable portion of the rib or ribs involved should be removed with it.

The resemblance of these growths during their early stages to chronic inflammatory swellings makes their early recognition difficult. The question can often be decided only by cutting down and removing a portion of the growth for microscopical examination. This investigation should always be made in cases where the suspicion of malignancy is strong, for if the swelling prove to be a sarcoma its early removal is imperative, while in chronic inflammatory conditions an incision is often beneficial rather than otherwise.

DISEASES

OF THE

THYROID AND THYMUS GLANDS.

By OLIVER P. REX, M.D.

DISEASES OF THE THYROID GLAND.

Despite many late investigations in regard to the matter, it must be confessed that we have no definite and conclusive knowledge as to the function of the thyroid gland. From numerous experiments upon animals, especially by Ewald, Schiff, Rogowitsch, and Horsley, it seems that herbivora bear extirpation of the gland much better than carnivora. In man its extirpation produces a state of cachexia strumipriva, or virtual cretinism. Most dogs, cats, etc., soon die, after a period of hebetude and lethargy. An extract of the thyroids injected subcutaneously into another dog produces similar lethal results. Cardone[1] believes that the thyroid and the spleen have physiological and pathological relations, and that the thyroid has evidently some hæmopoietic function. Horsley also regards it as a blood-forming organ, and says that during the anæmia following its removal the blood of the thyroid vein contains seven per cent. more red blood-corpuscles than the corresponding artery. It seems also to regulate the formation of mucin in the body. Horsley classes the symptoms following excision of the gland into three stages,—the neurotic, the mucinoid, and the atrophic. Young animals resist the changes much less than older ones. The general result so far seems to point to the thyroidic function as having a close relation with and as being of great importance to the central nervous system, probably in removing from the body certain products injurious to that system.

Absence of the thyroid is very seldom met with. Anomalies are more common, consisting of abnormal smallness or largeness, absence of a lobe or of the isthmus, and accessory glandular masses separate from the main mass. Four cases have been reported of congenital development of thyroid tissue inside the trachea.

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[1] Archivi Italiani di Laringologia, p. 4.

The diseases of the thyroid in children are hyperæmia, goitre, cretinism, exophthalmic goitre (rarely observed in children, and treated in vol. iv. of this work), and neoplasms.

HYPERÆMIA, OR THYROIDITIS.

Etiology.—Turgescence or swelling of the thyroid may be induced by bronchial or chest diseases, by valvular disease of the heart, by intermittent fever, and by local disturbances of the innervation of the vessels supplying the organ. Impure water is also charged with causing this as well as the more pronounced goitre. The cases reported as arising secondarily to or in connection with acute rheumatism, arthritis, ague, malaria, etc., are usually, if not always, in adults. Mechanical causes, such as excessive crying, singing, carrying heavy weights upon the head, compression of the neck, etc., are also adduced. Dr. Barlow[1] reports a case of severe acute enlargement of the thyroid in a child three years of age, the symptoms culminating in four days, with entire reinstatement of health and normality within about two weeks. It seems to have been a good example of the true idiopathic type of the disease; though the author mentions that the child was exposed to cold when barely recovered from an attack of erythema nodosum.

Symptomatology.—There will be moderate difficulty of breathing or noisy breathing, giddiness, and other evidences of venous cerebral congestion. One-sided flushing of the face has been noticed, and sometimes quickened and irregular action of the heart. The gland is swollen, the throat rounder and larger externally. In rare cases the course of the affection has been so acute and severe as to produce in a few hours such compression of the trachea or veins of the neck as to end in death. In most cases the constitutional symptoms are inconsiderable.

Treatment.—Change of residence and careful attention to sanitary regulations are demanded. In very acute cases it may be necessary to bleed, or to perform tracheotomy.

GOITRE.

Synonymes.—Guttur tumidum (Pliny); Hernia gutturis or Bronchocele (Paul of Ægina); Botium or Bocius (School of Salerno); Thyreophraxie (Alibert); Thyreophyma; Bronchocele and Hydrocele colli (names used a century ago); German, Kropfstruma; Italian, Gozzo, Bozzolo; Spanish, Papera; French, Goître, Grosse gorge, Gros cou; English, Wen, Derby neck.

Definition.—Goitre (from Latin *guttur*, the "throat") is a chronic hypertrophic enlargement of the thyroid gland.

Etiology.—The results of the labors[2] of the English committee appointed to investigate myxœdema render it probable that the diseases called myxœdema, cretinism, cachexia strumipriva, and goitre are, if not identical, at least different aspects or results of a common cause, with which the condition of the thyroid gland is intimately connected. The absence, disease, atrophy, or extirpation of the gland seems absolutely necessary for the rise of the pathological conditions mentioned. We are thus far in ignorance as to the ultimate cause that lies at the origin both of the glandular and of the other disease.

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[1] Clinical Society's Transactions, vol. xxi.

[2] Lancet, June 2, 1888.

Goitre may be congenital or acquired; it may be sporadic, endemic, or epidemic. Vetlesen[1] made a study of one hundred and seventeen families of Hamar, Norway, in each of which one or more members suffered from goitre, and he conclusively proves it to be largely of hereditary origin, and frequently intercurrent with other diseases of the vaso-motor system in other members of these families. It is thought that the vaso-motor origin of goitre is thus indirectly proved. Endemic goitre is often sharply limited from adjacent districts that are free from it. The influences of geology, climate, atmosphere, condition of the drinking-water, etc., have been credited with the production of goitre. Girls seem more subject to this disease than boys. It is not improbable that hyperæmia of the thyroid produced by the conditions already mentioned may serve to beget chronic goitre, but the local epidemic and endemic characters of the disease point to local conditions, and a very plausible theory is that some local exciting cause exists, of a miasmatic nature, independent of the altitude or temperature, but developing only over certain kinds of rock or soil. Bircher believes it occurs only on marine deposits of the palæozoic, triassic, or tertiary age. He thinks that endemic goitre and deaf-mutism, cretinism, and cretinoid idiocy are all due to the same miasm. That these conditions and those called cachexia strumipriva and myxœdema are intimately bound up with the function of the thyroid gland, there can no longer be any doubt. The more generally accepted belief is that the disease is due to the presence of lime and magnesia salts in the drinking-water. St. Lager finds that cretinism is confined to metalliferous districts where iron and copper pyrites abound. Morel, Virchow, and Koeberlé think it due to malaria, or even to a special organic germ. No explanation seems to be wholly satisfactory.

Pathological Anatomy.—The excess of tissue may consist either of an over-distention or of a telangiectatic dilatation of the vessels, hypertrophy of the gland-tissue, premature and excessive colloid deposit, increase of the fibrous stroma, or adenomatous growth. In children the enlargement of the thyroid of goitre seems little more than a continuation of the natural growth, a true hypertrophy or an excessive development of the normal tissues. The peculiar fibrous, cystic, or colloid abnormalities of the adult goitre are rarely present. There is commonly only a follicular or lymphatic hyperplasia of the parenchyma of the gland, with extreme vascularity. The adenomatous growth is divided by Wölfler into four varieties,—the fœtal, nodulous growths from embryonic tissue; the gelatinous, occupying the whole gland of a single lobe; the myxomatous, soft vascular nodose growths; and columnar-celled vessels or acini lined with tall columnar epithelium.

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[1] Journal of Laryngology, March, 1888.

The effects of goitre upon the brain, in the main, proceed from venous stasis and denutrition. An extreme case came under my care while I was an interne of the Philadelphia Hospital in 1867. I held a post-mortem examination on a child, a goitrous cretin, and found the ventricles of the brain enormously distended with serous effusion, with great thinning and atrophy of the cortical portion of the brain,—the whole evidently a result of pressure and venous stasis due to the goitre.

Acute inflammation, suppuration, hemorrhage, or encysting of goitres is frequently met with in adults, but hardly ever in children. A congenital form called varicose goitre is found, in which the dilated veins become thin and sacculated. More infrequently aneurismal dilatations of the arteries are found, in which, by the anastomoses of small aneurisms, a pulsatile, erectile tumor is formed.

Symptomatology.—The right side is usually larger than the left. Sometimes a hyperæmia precedes the acquired goitre, but it generally arises without noticeable thyroiditis. It may grow with great rapidity or with extreme slowness. The subjective symptoms will depend on its relations with the organs of the neighborhood and upon the amount and direction of its pressure upon them. The most serious symptoms arise from this pressure upon the trachea, the œsophagus, or the vessels of this region. When the goitrous growth extends beneath the sternum, the trachea becomes necessarily compressed; this also may occur when the goitre becomes large and surrounds the trachea or pushes it aside. The accessory thyroid masses may become goitrous just the same as the principal gland. The age of the child, natural tenseness of the tissues, shape of the neck, etc., will modify these phenomena. There is venous stasis, first of the goitre itself, then of the cerebral vessels, with the usual cerebral symptoms, drowsiness, giddiness, headache, etc. Cerebral anæmia from compression of the carotids more rarely happens. There may be ringing in the ears and defects of hearing. There is more or less difficulty of breathing, from compression of the trachea and (rarely) of the larynx. From the compression of nerves there may result aphonia, loud and piping breath-sounds, changes of voice, etc. Swallowing is sometimes rendered difficult by pressure upon the œsophagus. In the epidemics of goitre of European countries the disease runs a rapid course, from eight to twenty days, when the enlargement of the neck disappears, sometimes leaving a slight chronic swelling.

Diagnosis.—The differential diagnosis between sarcomata and lymphomata, ranulæ, and dermoid cysts may present difficulties, but such cases are comparatively so rare that we need not discuss the subject here. In cystic goitre, the fluctuation of the tumor, or more certainly the passing of a small trocar, will quickly clear up the diagnosis.

Prognosis.—Spontaneous cures frequently take place. Cases where the growth is superficial and of slow increase have a good prognosis. The rarer cystic deep-lying or fibrous varieties are of doubtful prognosis.

Treatment.—It is highly important that those predisposed, or in whom the tendency to goitre has already shown itself, should speedily be removed to another locality. Alkaline mineral waters are of undoubted efficacy. Otherwise they should drink only such water as has been boiled, have well-ventilated rooms, and avoid all such bodily exertion as tends to bring an increased blood-pressure about the neck. Long labors in childbirth, with the consequent circulatory disturbances, have been credited with producing goitre or favoring its development. Iodine internally and locally is an old remedy and a good one. Probably the most general and successful treatment of fibroid goitre is by hypodermic injections of the tincture of iodine deeply into the substance of the tumor. A small amount, say a half-syringeful, should be injected at first until the effect is observed. This may be repeated about once a week, the quantity injected being regulated by the age of the patient, by the reactions both local and systemic, and by the size of the tumor. In large bronchoceles the treatment may have to be kept up for many months. In small ones a few injections are sufficient to reduce the parts to normality. Great care must be taken to avoid the blood-vessels in inserting the needle. Ligation of one or more of the thyroid arteries has been advocated, and in some cases has been successful, but the proved danger of myxœdema from any surgical interference with the gland must render the greatest caution necessary. Some authors still practise partial or even complete extirpation of the gland, and report good results therefrom, but the general trend of opinion is against it, owing to the frequency of resultant cachexia strumipriva. When other plans of treatment have failed, and when the injury is so great that this dangerous operation is advisable owing to the danger to life from further non-interference, then it is of course well to proceed with either partial or complete extirpation of the gland. Ligation of the arteries preceding excision of the gland has been found of advantage, at least in rendering the operation bloodless. For cystic goitre the usual treatment is drawing off the fluid with a trocar and canula and then refilling the cyst with a solution of iron (tincture ʒii to water ℥i). This proceeding requires care not to inject air into a vein.

CRETINISM.

For a complete account of this subject we refer the reader to the article by Dr. Bury in this volume.

NEOPLASMS.

Cases of carcinoma and of sarcoma of the thyroid, and also of syphilis and tuberculosis, have been reported as occurring in children. Owing to the difficulty of the differential diagnosis between these and goitre, perhaps many have escaped notice.[1]

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[1] See the article Affections of the Mediastinum, in this volume.

DISEASES OF THE THYMUS GLAND.

The thymus gland develops from the seventh week of fœtal life and during the first two or three years after birth; it then remains stationary until the tenth, twelfth, or fourteenth year, when it rapidly undergoes fatty degeneration and atrophies, so that by the twentieth year only a trace of it remains. At birth it weighs from one hundred to two hundred grains. While functional it seems to be a true lymph-gland,—a view confirmed by the fact of its remaining through life an active organ in those animals which do not have lymph-glands. From experiments upon animals, its extirpation seems to be followed by no considerable result. In leucocythæmia and lymphadenoma this gland, like others, is enlarged.

Instances of its abnormal size without any apparent or considerable result upon the health have been reported,—one case where it weighed over six hundred grains. Other cases of its prolonged existence have been recorded.

Owing to its position, our comparative ignorance of its function, and the doubtfulness of the differential diagnosis between diseases of the thymus and those of neighboring structures, comparatively few cases of certain thymic diseases in children have been reported. Friedleben's large work[1] gathers the knowledge of the subject to his time, and since then Somma,[2] Sanné,[3] and Jacobi[4] have contributed noteworthy articles upon the subject. To the last work the writer acknowledges especial indebtedness: of the thirty-two cases examined by Jacobi there were four cases of tuberculosis, five of syphilis, and a number of diphtheria. There was one case of persistent thymus.

THYMIC ASTHMA.

Kopp was the first to advance the hypothesis of respiratory disturbances due to the compression of an hypertrophied thymus: many have held the inference unjustifiable, among whom are Rilliet, Barthez, and Friedleben, but West, Sanné, Grawitz, Clar, Goodhart, and Jacobi accept it. Jacobi points out that the distance in an infant of eight months between the manubrium sterni and the vertebral column amounts to but four-fifths of an inch; whence it appears that compression may result from congestion or hypertrophy of the thymus, and that sudden death may result from this cause. Grawitz[5] reports two such cases, in one of which the nurse was charged with criminal neglect, and in the other the child died suddenly in apparently good health; in both cases the necropsy showed extreme enlargement of the thymus. Other cases are reported by Goodhart[1] and Clar.[2] Among many cases of laryngismus stridulus Jacobi has met with a dozen deaths, and in one of these twelve the sudden death is attributed to the size of the thymus gland. Erb notices that enlarged thymus and thyroid coexist with acromegalia, or morbid giant growth.

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[1] Die Physiologie der Thymusdruse, etc., Frankfurt-am-Main, 1858.

[2] Sulla Tracheostenosi, etc., Arch. di Patol. Inf., 84.

[3] Dict. encyclop. des Sci. méd., 1887, art. "Thymus."

[4] Contributions to the Anatomy and Pathology of the Thymus Gland, Trans. Assoc. Amer. Phys., 1888.

[5] Deutsche Med. Wochenschr., No. 20, 1888.

SUNDRY AFFECTIONS OF THE THYMUS GLAND.

Demme[3] reports a case of isolated primary tuberculosis of the thymus in a new-born child of non-tuberculous parents. In all the cases of Jacobi the bacillus tuberculosis was present, the tuberculous tissue appearing in the thymus as an infiltration of the organ with spheroidal or polygonal cells held together by a delicate basement-substance without characteristic arrangement, the arteries being the seat of obliterating processes.

The cases of so-called suppuration of the gland are possibly and even probably to be excluded, on the ground that the liquid contents of the normal gland are in color, consistency, etc., not unlike pus. Several cases of syphilitic changes of the thymus are reported in fœtuses or children of syphilitic parentage. In two hundred autopsies of infants of congenital syphilis Furth found seven cases of what he claimed to be syphilitic thymus, showing the characteristic changes of the blood-vessels; other cases of hemorrhages into the thymus coincident with congenital syphilis have been reported. Jacobi especially notices the excessive amount of connective tissue in the thymuses of syphilitic infants. Whether the thymus were large or small, the changes in the blood-vessels showed a general thickening of all the coats.

A number of cases of malignant tumors of the thymus have been reported,—a hemorrhagic sarcoma by Steudener,[4] a lymphosarcoma by Grutzner,[5] a lymphadenoma by Rosenberg,[6] and another lymphosarcoma by Ballag.[7] Cases of enlarged thymus in leucocythæmia have also been reported. In all such cases the diagnosis during life is of extreme difficulty. Extensive dulness over the manubrium sterni, with any symptoms of compression of the thoracic viscera, will of course put one on his guard.

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[1] British Medical Journal, 1879.

[2] Jahrbuch für Kinderheilkunde, 1858.

[3] Twenty-Second Report of the Children's Hospital in Bern, 1885.

[4] Virchow's Archiv, 1874, lix. 463.

[5] Inaug. Dissert., Berlin, 1869.

[6] Inaug. Dissert., Göttingen, 1884.

[7] Inaug. Dissert., Brugg, 1887.

AFFECTIONS OF THE MEDIASTINUM.[1]

BY WILLIAM A. EDWARDS, M.D.

CARCINOMA OF THE MEDIASTINUM.

CARCINOMA of the mediastinum is not a frequent disease in childhood, although the literature presents a number of cases.[2] (See Table I.)

Upon analyzing the eleven cases of mediastinal carcinoma in children, we find that the youngest was four years of age and the oldest eighteen; three occurred at twelve years of age, two at eleven, and two at fifteen, showing that between the eleventh and the sixteenth year carcinoma of the mediastinum is most apt to develop in children, and that the male sex in the early periods of life is most liable to the growth of the neoplasm, as of these eleven cases eight were males. In regard to the area involved, we find that the anterior mediastinum was affected alone six times, the anterior and the posterior mediastinum four times, and the "whole left side" once, the growth in this instance having its primary seat in the mediastinum. The duration of the disease was in the longest instance three years, and in the shortest one month,—all the cases resulting fatally.

SARCOMA OF THE MEDIASTINUM.

The literature presents sixteen cases of sarcoma in this situation in childhood, ranging between the ages of five and eighteen years, which we shall proceed to analyze. (See Table II.)

These sixteen cases demonstrate the very interesting fact—one that is worthy of remembrance—that sarcoma of the mediastinum is more frequent in childhood than carcinoma of the same structure. Just the converse of this proposition is true of adults, in whom carcinoma is most frequent in this situation.

Of the recorded cases of the two diseases, sarcoma has almost a thirty-per-cent. higher rate of occurrence than carcinoma; the former is also more apt to arise at an earlier age, as twenty-five per cent. of the cases occurred at eight years of age, and twenty per cent. of the cases of carcinoma occurred at twelve years of age, the period of life in the young at which this disease is most usually manifested.

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[1] In the preparation of this article I have availed myself of my recent publications upon this subject which have appeared in the *Archives of Pediatrics* and in the *Occidental Medical Times*, both for July, 1889.

[2] For these and other cases referred to I am indebted to Dr. Hobart A. Hare, Fothergillian Essay, 1888,—the most recent and most complete *résumé* of the subject.

Of these cases nine affected the anterior mediastinum alone, two affected the anterior and posterior mediastinum alone, one affected the posterior mediastinum alone, two affected the "entire" mediastinum, one affected the "sternum," and one affected the "whole thorax."

As appears to be usual in childhood, the anterior mediastinum was affected most frequently: nine out of the sixteen cases occurred in this locality. In this respect the child resembles the adult, as carcinoma and sarcoma are most frequently seen in the anterior mediastinum.

Sex seems to have a marked relation to the occurrence of the disease, as two-thirds of the recorded cases were males. This is certainly a striking preponderance of the male sex, and whether it is due to the meagre number of cases upon which to base conclusions of course can be decided only when our literature shall have become more voluminous: however, almost the same relation between the sexes is recorded in carcinomatous deposits, as eight of the eleven cases were males; and the same is true of adults, the males suffering much more frequently than the females.

It is worthy of record that sarcoma is so frequently primary in the mediastinum, as we are accustomed to consider it a growth most liable to metastasis, and in post-mortem examinations of sarcomatous deposits it is most usual to find numerous foci of metastatic deposit; but when sarcoma is deposited in the mediastinal tissues it seems to have a tendency to remain local, as in these sixteen cases it arose in twelve within the mediastinum, and in twelve of them remained almost within the structure throughout its growth, except in two instances, where some extension into the lung-parenchyma is noted; in the remaining four cases the original observers failed to state whether it had its primary origin within the mediastinum or elsewhere. The records show, as one would suppose, that, owing to the richness of lymphatic tissues in this situation, sooner or later the middle and posterior spaces become affected also.

Dr. Angel Money showed to the Pathological Society of London a specimen of mediastinal sarcoma in an infant aged fifteen months. It was the size of a man's fist, and projected chiefly into the right side of the thorax. It was one-fourth the size of the thoracic cavity, and caused extensive collapse of the lungs. It pushed the heart, aorta, and vena cava in front of it, and displaced the liver downward. It did not grow from the vertebræ, and the spinal column was not eroded. During life the symptoms resembled those found in extensive collapse of the lung; the physical signs were extreme dulness of the right lower half of the chest, with absence of breath-sounds; elsewhere bronchitic râles obtained. An exploring-needle thrust into the dull area felt as if held in a dense solid tissue; no fluid could be withdrawn. Microscopic examination proved the tumor to be a round-celled sarcoma without any striated muscular tissue.[1]

In regard to the variety of growth which is most frequently met with, our series shows that lymphosarcoma occurred ten times, round-celled sarcoma three times, and in three cases the variety was not mentioned: this again is analogous to the adult, in that the greatest number are classed as lymphosarcoma. No cases of spindle-celled sarcoma are recorded in the child.

MEDIASTINITIS.

Abscess or *suppurative mediastinitis* is a not infrequent disorder of childhood; eighteen cases are recorded under eighteen years of age in a total of one hundred and fifteen of all ages, and of the latter ten are noted out of sixteen cases of all ages. (See Tables III. and IV.)

Males are far more prone to be affected by mediastinal abscess than females, in the proportion of fourteen to two (in two instances the sex was not stated); the youngest was aged three and a half months, the oldest eighteen years. From the sixteenth to the eighteenth year is the period of life among the young during which abscess will be most likely to arise.

Of these eighteen cases, two were tubercular, one was scrofulous, five were due to trauma, two were cold, three acute, two metastatic, one is recorded as a congestive abscess, and of two the variety is not stated. In ten cases the abscess occurred in the anterior mediastinum; in four, in the posterior mediastinum; in three, in the mediastinum (?); in one case, in the middle mediastinum. Of the first ten cases of abscess five were due to trauma, which is readily understood, owing to the exposed situation of the anterior mediastinum, which is so accessible to traumatic injuries resulting in mediastinitis and abscess; indeed, all the traumatic cases recorded are situated in the anterior mediastinum. In looking over the other etiological factors in the production of abscess we note one case following broncho-pneumonia, two tracheotomy, one a metal pin in the throat, another as a result of erysipelas; Daudé regards rheumatism as a primary factor in his case; another was a concomitant of caseous bronchial glands, and two were tubercular.

Hare considers that the exanthemata, particularly measles and typhoid fever, demand attention as causative factors in the production of abscess in the region under consideration; but we are unable to find any cases in childhood in which this relation has been established.

The *duration* of an abscess is very uncertain, depending upon the variety, and ranges from six or seven hours after the first symptoms noted to a period of nineteen years (chronic abscess). Most of the acute cases, however, run a short course, usually terminating in death. Five recoveries are recorded in eighteen cases of all varieties.

Cold abscess is not so frequent among the young as in adults, in whom the proportion is thirty-one of the cold to forty-eight of the acute variety. In childhood it is much smaller,—fourteen acute to three chronic cold abscesses.

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[1] Archives of Pediatrics, July, 1889, p. 494 (Brit. Med. Jour., November 10, 1888).

Simple or non-suppurative mediastinitis presents but two cases which come within the allotted age of the present study. Both cases were males, and about the same age,—ten years. The average age at which adults are affected is about twenty years. One of these cases was associated with pericarditis, and the other appears to have been part of a general process involving the glandular structures of the mediastinum; both cases were fatal, one in a short time, the other within fifteen months.

The number of cases is so small that of course we can draw but few if any conclusions; trauma is not mentioned, and in these two instances, at least, suppuration did not arise, although it is the most usual termination.

LYMPHOMA AND LYMPHADENOMA.

We shall not enter upon a discussion as to the relation of lymphoma or lymphadenomatous growths to sarcomata, nor as to the relative malignancy of the two. Suffice it to say that much confusion exists in regard to these matters, and we shall for the present be obliged to content ourselves with the statement that in some instances lymphadenoma is extremely malignant and in others equally benign, and that lymphoma more frequently manifests the latter characteristic than it does the former. (See Table V.)

Again, it is to be noted that males are affected in the proportion of three to one, and that the deposit occurred in the anterior mediastinum twice, and in the posterior and the entire mediastinum once each.

DISEASES OF THE GLANDS.

The glands in the mediastinum often become enlarged, hyperæmic, and indurated, and cause disturbances either from pressure or from the formation of abscess and the burrowing of pus. Goodhart has recorded four instances of enlarged mediastinal glands in children from eight months to two and one-half years of age. Gravenhorst adds a case of very large tubercular glands in the middle and posterior mediastinum, which caused death from pressure. In the *Edinburgh Medical Journal*, 1848 (quoted by Hare), is recorded a case in which a foreign body penetrated the middle mediastinum in a child between five and six years of age, making an opening five inches deep between the œsophagus and the trachea, and communicating with the trachea. The case resulted fatally within a few days.

Eberth[1] records an instance of what he calls "mycotic" mediastinitis, in a boy aged seven, affecting principally the posterior mediastinum; Baseri,[2] an example of tubercular enlargement of the mediastinal glands in a child who also presented a cavity in the right lung; Jones,[3] a cystic tumor in the anterior mediastinum in a boy aged nine, which was said to have followed a blow on the chest; and Rich and Bowen, a case of pyopericardium, accompanied by a pulsating tumor of the anterior mediastinum.[1] Goetz[2] records a tumor of unknown variety which occupied the entire mediastinum in a girl aged fifteen; and Wilkes,[3] a "lardaceous" deposit in the anterior and posterior mediastinum, in a lad aged eighteen. This case resulted fatally, after a duration of one year.

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[1] Deutsch. Arch. f. Klin. Med., Bd. xxviii. Hft. 1.

[2] Jahrb. f. Kinderkrankheiten, 1878, vol. xii. p. 415.

[3] Brit. Med. Journal, 1880, vol. i. p. 286.

MISCELLANEOUS DISEASES OF THE MEDIASTINUM.

Following Hare's example, we shall now consider some cases which, for various reasons, can be considered only under the above head. (See Table VI.)

No cases of *fibroma, lipoma, hæmatoma,* or *dermoid cysts* are recorded in individuals whose age would allow of a consideration in this article, and but one case of *hydatid cyst* (echinococci) of the mediastinum is to be found in the literature; this occurred in a male aged eighteen, and involved the mediastinum (entire?), also affecting the lungs and intestinal tract. The chief symptoms noted were cough, remittent fever, quick respiration; the duration is not stated, but the patient succumbed to the disease. The presence of echinococci was demonstrated.[4]

Age is no factor in the consideration of hydatid disease of the mediastinum, because, no matter what the period of life may be, should the eggs gain entrance to the body, development of the disease will of course follow.

GENERAL SYMPTOMATOLOGY OF MEDIASTINAL DISEASE.

The various diseases of the mediastinum closely resemble one another in their symptomatic manifestations: indeed, let the cause be what it may, they all have certain symptoms in common. This is only what we should suppose when the nature of the structure is considered, as all growths must interfere with the tissues or organs contained in one or other of the mediastini, and evidences of pressure on either the circulatory or the respiratory apparatus are noted in almost all cases; indeed, dyspnœa is an almost invariable concomitant of mediastinal disease, cyanosis is almost as frequent, and pain is a constant symptom, particularly in acute abscess, and in sarcomatous and carcinomatous deposits; in the two latter dysphagia often becomes an alarming element in the case.

In acute and sometimes in chronic abscess flushes of heat or rigors may be noted, more particularly, of course, in the former; pulsation may be evident not alone to medical observers but also to the patient, and the abscess may appear externally, when the differential diagnosis between this condition and aneurism must be made.

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[1] Liverpool Med. and Chir. Jour., 1882, vol. ii. p. 344.

[2] Berlin. Klin. Wochenschr., 1885, vol. xxii. p. 83.

[3] Trans. Path. Soc. Lond., vol. x. p. 259.

[4] Gueterbock, Deutsches Zeitschrift f. Klin. Med., vol. xx. p. 82

Pain, to a greater or less extent, is always present; its intensity, of course, depends upon the tissues pressed upon or incarcerated in the new growth; in some instances it remains localized, in others it extends over the thorax, up the neck, and down one or other arm.

The cough is somewhat peculiar, and is similar in character to that which has been associated with aneurism; indeed, as the cause of the cough in mediastinal disease is probably identical with that of the cough in aneurism,—*i.e.*, pressure,—it is only natural to find them very similar in tone, intensity, and degree.

Much emaciation is always observed, and in the malignant cases a marked cachexia arises very early in the course of the disease; the pupils are not infrequently irregularly dilated or contracted; the cervical, post-cervical, and occipital glands become hyperæmic, indurated, and enlarged.

TABLE I.—CARCINOMA OF THE MEDIASTINUM.

AGE.	SEX.	AREA INVOLVED.	OTHER PARTS AFFECTED.	CHIEF SYMPTOMS.	DURATION.	RESULT.	BY WHOM AND WHERE REPORTED.	VARIETY.	PRIMARY SEAT.
4	M.	Anterior mediastinum.	All the abdominal contents.	Anorexia and wasting.	A few weeks.	Death.	C. Ferrall, Dublin Jour. Med. Sci., Aug. and Nov., 1816, p. 510.	Encephaloid.	Not stated.
11	F.	Whole left side.	Whole left side.	Dyspnœa and lividity of face.	Not stated.	Death.	Bennett, Intrathoracic Growths, London, 1872, p 101.	Medullary.	Mediastinum.
11	M.	Anterior and posterior mediastinum, extended from middle dorsal region to sacrum.	Muscles of back, dura mater of cord, pleura, and lungs, all were cancerous; cancerous glands in groin; left ureter occluded by cancer of wall of bladder pressing on it.	Great anæmia, swollen belly, and tympanites; numbness of left arm, with œdema of left leg and thigh.	About 2 months.	Death.	Bennett, ibid., p. 137.	Encephaloid.	Not stated.
12	F.	Anterior and posterior mediastinum.	Right pleura and lung; heart.	Pain, cough, and dyspnœa.	1 month.	Death.	Burton, Med. Times and Gazette, Sept. 4, 1880, p. 266.	Lymphoid cancer.	Mediastinum.
12	M.	"There existed no mediastinum, neither posticum nor anticum, but this, that the pleura came together, and their walls were found attached not only to the lungs but the ribs."	....	Cough, pain, and dyspnœa.	Not clearly stated.	Death.	Hufland, Jour. der Praktischen Arzneikunde, xxv. 187.	Scirrhus.	Not stated.

12	F.	Anterior and posterior mediastinum.	Pleura, lungs, thoracic gland, and kidney.	Cough, pain, and dyspnœa.	Several years.	Death.	Gross, Phila. Med. Times, 1879, ix. 291.	Scirrhus.	Breast.
15	M.	Anterior mediastinum.	Lung, blood-vessels, and bronchi; also pericardium.	Dyspnœa, dry cough, pain in chest.	3 or 4 months.	Death.	Wunderlich, Handbuch der Path. und Ther., iii., Bd. 2, p. 673.	A white cartilaginous mass	Not stated.
15	M.	Anterior mediastinum; entire right side of chest.	Eroded upper ribs; adherent to sternum and costal pleura.	Dyspnœa, croupy, cracked cough; pain between scapulæ.	About 3 months.	Death.	Burrows, Medical Times and Gaz., June 7, 1851; also London Jour. of Med., July, 1851.	Soft cancer.	Not stated.
16	M.	Anterior mediastinum.	Kidneys and right lung.	Dysphagia and dulness on percussion on right side; dyspnœa.	1 month.	Death.	Barker, Trans. Path. Soc., London, vol. vii. p. 45.	Not stated.	Mediastinum.
17	M.	Anterior mediastinum.	Attached to upper surface of diaphragm; surrounded trachea and roots of lungs; adherent behind the chest-wall.	Dyspnœa; dulness over entire side on the left; cyanosis.	Seen 9 weeks.	Death.	Thompson, Medical Mirror, London, 1865.	Scirrhus.	Mediastinum.
18	M.	Anterior mediastinum.	Pressed on bronchus and displaced heart; adherent to pericardium.	Dyspnœa and pain; tumor pulsated.	3 weeks after appearance of tumor.	Death.	Nélaton, Bull. de la Soc. Anat., 1833, p. 105.	Not stated.	Not stated.

TABLE II.—SARCOMA OF THE MEDIASTINUM.

AGE.	SEX.	AREA INVOLVED.	OTHER PARTS AFFECTED.	CHIEF SYMPTOMS.	DURATION.	RESULT.	BY WHOM AND WHERE REPORTED.	VARIETY.	PRIMARY SEAT.
5	F.	Anterior mediastinum; extended from sternal notch to diaphragm.	Enclosed pericardium and heart on all sides except the back; glands in posterior mediastinum enlarged.	Cachexia; œdema of face; purpura hæmorrhagica; spleen enlarged; low bodily temperature.	3½ months.	Death.	Gamgee, Edin. Med. Jour., March, 1873, p. 797.	Lympho-sarcoma.	Thymus.
5	M.	Anterior mediastinum.	Attached to sternum and upper part of pericardium; superior vena cava thickened.	Cyanosis; loss of flesh; vocal fremitus impaired.	10 months.	Death.	Cobet, Inaug. Dissertation, Marburg, 1870.	Lympho-sarcoma.	Mediastinum.
8	M.	Mediastinum.	Growth invaded lungs along bronchi; infiltrated pericardium and upper part of auricles, also glands of mediastinum.	Dyspnœa mainly expiratory.	Over 2 months.	Death.	Hutton, Brit. Med. Jour., 1887, vol. i. p. 735.	Not stated.	Not stated; probably mediastinal.
8	M.	Anterior mediastinum.	Involved lungs and pericardium; pressed on trachea.	Cough, dyspnœa; cyanosis; veins of right side of face full.	2 or 3 years.	Death.	Grützner, Dissertation, Berlin, 1869.	Lympho-sarcoma.	Mediastinum.
8	M.	Whole thorax.	Affected pericardium and spread into lungs, along vessels and bronchi.	Not stated.	Urgent symptoms lasted 8 weeks.	Death.	Hutton, Lancet, Lond., April 30, 1887, p. 883.	Not stated.	Glands of mediastinum.
8	M.	Anterior mediastinum.	Edges of right lung; vagus and pulmonary artery involved.	Cough, dyspnœa, cyanosis, and swelling of glands; pain in chest.	About 5½ months.	Death.	Rosenberg, Ueber Mediastinal-Tumoren bei Kindern, Göttingen, 1884.	Lympho-sarcoma.	Thymus or gland of mediastinum.
9	F.	Anterior and posterior mediastinum.	Sternum, ilium, sacrum, and vertebræ.	Palsy of lower extremities; wasting.	3½ months.	Death.	Jones, St. Barthol. Hospital Reports, 1884, vol. xx. p. 225.	Round-celled sarcoma.	Not stated.
9	F.	Sternum.	All vertebræ below the fourth dorsal.	Palsy of lower extremities; sensation partly lost; incontinence of urine and fæces.	40 days.	Death.	St. Barthol. Hosp. Reports, vol. xx. p. 225.	Lympho-sarcoma.	Not known.

11½	M.	Entire mediastinum.	Involved pericardium.	Cough; pain in chest.	3 months.	Death.	Huber, Deutsch. Arch. f. Klin. Med., xvii. 496.	Lympho-sarcoma.	Not stated.
14	M.	Anterior and posterior mediastinum.	Adherent to sternum, ribs, and diaphragm; attached to pericardium; pleural cavities contained much fluid.	Dyspnœa; blueness of face; jugulars swollen.	3 weeks.	Death.	Cole, Lancet, Lond., Oct. 23, 1875, p. 586.	Lympho-sarcoma.	Not stated.
14	M.	Anterior mediastinum.	Compressed trachea; enlarged veins of chest, particularly the superior vena cava; implicated the œsophagus and pericardium at base.	Asphyxia; signs of tracheal stenosis; neuralgic pains in left arm and shoulder; enlarged glands above clavicle; œdema and cyanosis of face.	9 months.	Death.	Wiss, Proc. 9th Internat. Med. Cong., 1887, Sect. Dis. of Chil.	Lympho-sarcoma.	Thymus gland.
15	M.	Anterior mediastinum.	Brachial plexus and all the vessels on the left side, subclavian, carotid, jugular, and innominate, were included in the growth.	Cough; pain in left arm; rigors and night-sweats.	2½ months.	Death.	West, Trans. Path. Soc., Lond., 1883, vol. xxxiv. p. 233.	Round-celled.	Anterior mediastinum.
15	M.	Chiefly in posterior mediastinum.	Pressed on lung; œsophagus involved; left arm œdematous.	Left pupil contracted; pain in chest; œdema.	5 months.	Death.	Smith, Brit. Med. Jour., Dec. 30, 1876, p. 869.	Lympho-sarcoma.	Mediastinum.
16	F.	Anterior mediastinum; reached from thyroid to diaphragm.	Pericardium involved; pleura adherent to sternum and ribs.	Dyspnœa and enlargement of cervical veins.	3 months.	Death.	Allbutt, Brit. Med. Jour., Sept. 5. 1874, p. 300; also Rev. des Sci. Médicales, vol. v. p. 530.	Not stated.	Mediastinum.
17	M.	Anterior mediastinum.	Metastasis to heart-muscle and both kidneys.	Cough, fever, anorexia, and cyanosis.	25 days.	Death.	Schreiber, Deutsch. Arch. f. Klin. Med., xxvii. 52.	Round-celled sarcoma.	Anterior mediastinum.
18	M.	Anterior mediastinum.	Pericardium, aorta, and trachea were compressed.	Dyspnœa.	2 months.	Death.	Gee (reported by Moore), Trans. Path. Soc., Lond., xxxv. 374.	Lympho-sarcoma.	Anterior mediastinum.

TABLE III.—ABSCESS.

AGE.	SEX.	AREA INVOLVED.	OTHER PARTS AFFECTED.	CHIEF SYMPTOMS.	DURATION.	RESULT.	BY WHOM AND WHERE REPORTED.	VARIETY.	PRIMARY SEAT.
3 mos.	....	Posterior mediastinum.	Pressed on trachea.	Sudden dyspnœa; lividity of face.	6 or 7 hours after first symptoms.	Death.	Turner, Lancet, Lond., 1887, i. 17.		
5½ mos.	M.	Glands of anterior mediastinum.	Lungs studded with tubercle.	Cough and dyspnœa.	3½ months.	Death.	Ballard, Trans. Path. Soc., Lond., ix. 38, also Lancet, Feb. 6, 1858, p. 149.	Tuberculous abscess.	
18 mos.	M.	Suppurating tuberculous mediastinal glands.	Opened externally at suprasternal notch and right second interspace.	Wasting and night-sweats.	4 months.	Death.	Smith and Lankester, Med. Times and Gaz., Oct. 18, 1884, p. 539.	Tubercular.	
4 yrs.	M.	Posterior mediastinum.	Abscess reached from sixth cervical to fifth dorsal vertebra.	Dyspnœa and quick respiration.	Short, — not stated.	Death.	Jarisch, Jahrbüch. f. Kinderheilkunde, Jahrg. viii. Oct. 3, 1874, p. 188, also Rev. des Sci. Méd., vol. v. p. 609.	"Congestive abscess."	Due to broncho-pneumonia.
5½	F.	Anterior mediastinum.	Gangrene of edge of wound.	Skin livid; rapid pulse.	6 days.	Death.	Martini, Schmidt's Jahrbücher, vol. cii. p. 91.	Traumatic.	Emphysema following tracheotomy.
8	M.	Glands of posterior mediastinum.	Gland on right side was greatly enlarged, caseous and suppurating; vagi adherent to it; broncho-pneumonia and pleurisy of both bases.	Dyspnœa and cough; vomiting.	About 15 days.	Death.	Goodhart, Brit. Med. Jour., April 12, 1879.	Cold (?) abscess.	
Young lad.	M.	Anterior mediastinum.	Pyopericardium.	Great thirst; pulsating tumor of sternum.	1½ months.	Death.	Rich and Bowen, Liverpool Med. and Chir. Journal, 1882, ii. 341.	Acute abscess.	
11	M.	Posterior mediastinum.	Opening from mediastinum into pharynx.	Pains in chest; pus in expectorated fluid.	9 days.	Recovery.	See Bull. de la Soc. de Chirurg., 1875, N. S., i. 271.	Acute.	Metal pen in throat.
Boy.	M.	Anterior mediastinum.	Caries of sternum.	Not stated.	19 years.	Recovery.	Daudé, Les Affections du Médiastin, Paris, 1872.	Traumatic. cold. (?)	Fracture of ribs.

13	M.	Anterior mediastinum.	Separation of sternal fragments and pulsating tumor between fragments.	Symptoms simulating aneurism.	Not stated.	Recovery.	Smith, quoting Warner, "Cases in Surgery," Amer. Jour. Med. Sci., April, 1873, p. 311.	Traumatic.	Fracture of sternum; abscess burst externally.
Boy.	M.	Mediastinal glands.	Opened into trachea with escape of pus.	Sudden dyspnœa while at play; cyanosis.	Short.	Death.	Johnson, Brit. Med. Jour., Oct. 27, 1877, p. 592.	Probably scrofulous.	Had caseous bronchial glands.
16	F.	Anterior mediastinum.	Not stated.	Malnutrition, chill, fever, and redness of right knee.	5 months.	Recovery.	Daudé, Les Affections du Médiastin, Paris, 1872, p. 57.	Metastatic.	Rheumatism said to be primary cause.
16	M.	Mediastinum.	Not stated.	Fever, chills, and pain in chest.	…………………	Recovery.	Weber, Zeitsch. f. Med. Chir. und Geburtsh., Magdeburg, 1856, x. 58.	Cold abscess.	Mediastinum.
17	M.	Middle mediastinum.	Pressed on vagi; lungs engorged.	Pain; dyspnœa and cardiac palpitation; disturbed respiratory movements.	…………………	Death.	Winsor, Bost. Med. and Surg. Jour., 1867, lxxxvi. 63.	Metastatic.	Face and upper lip. Erysipelas.
17	M.	Anterior mediastinum.	Sternum contused and burnt.	Pain.	5 months.	Recovery.	Walker, Brit. Med. Jour., p. 63, Jan. 12, 1884.	Traumatic.	Struck in chest by red-hot iron.
Child.	………	Anterior mediastinum.	Tissue behind trachea.	Pain, oppression, and lividity of face.	9 days.	Death.	Martini. Schmidt's Jahrbücher, vol. cii. p. 91.	Traumatic.	Emphysema after tracheotomy for croup.
18	M.	Anterior mediastinum.	Luxation of manubrium and ensiform cartilages; third rib separated from sternum; lungs and pleura adherent to pericardium, which contained serum.	Marasmus.	Some months.	Death.	Duncan Reid, Annales de Schmidt, 1835, vol. i.		
18	M.	Anterior mediastinum.	Opened between third and fourth ribs; sternum carious: pericarditis; pleura and lungs slightly inflamed.	Pain in chest and left shoulder-blade.	Not stated.	Death.	Pfeufer, Henle's und Pfeufer's Zeitschrift, 1, 2; also Schmidt's Jahrb., Sept. 4, p. 273.	Mediastinitis resulting in abscess.	Scrofulosis.

TABLE IV.—MEDIASTINITIS—NON-SUPPURATIVE.

AGE.	SEX.	AREA INVOLVED.	OTHER PARTS AFFECTED.	CHIEF SYMPTOMS.	DURATION.	RESULT.	BY WHOM AND WHERE REPORTED.	VARIETY.	PRIMARY SEAT.
9	M.	Pericardium and mediastinum.	Thickening of bronchi; increase of fibrous tissues in lungs; hepatitis.	Not stated.	15 months.	Death.	Hutton, Brit. Med. Jour., March 18, 1881, p. 462.	Mediastino-pericarditis.	Pericardium and mediastinum.
10	M.	Middle mediastinum particularly.	Glands about trachea matted together, involving the large blood-vessels; adherent to pericardium.	Face and abdomen swollen; veins on chest prominent.	A short time.	Death.	Abstract of Med. and Surg. Cases, General Hospital for Sick Children, 1883, Pendlebury, Manchester, 1884.	Mediastinitis.	Mediastinum.

TABLE V.—LYMPHOMA AND LYMPHADENOMA.

AGE	SEX.	AREA INVOLVED.	OTHER PARTS AFFECTED.	CHIEF SYMPTOMS.	DURATION.	RESULT.	BY WHOM AND WHERE REPORTED.	VARIETY.	PRIMARY SEAT.
5	M.	Entire mediastinum.	Double pleurisy; trachea and bronchi involved.	Dyspnœa and cyanosis.	4 months.	Death.	Rosenberg, Beiträge zur Casuistik der Mediastinal-Tumoren bei Kindern.	Malignant lymphadenoma (probably a sarcoma).	
6	M.	Anterior mediastinum.	Tissues to left of sternum infiltrated by a yellowish mass.	Rapid breathing; dyspnœa; glands of neck enlarged; pain in chest.	5 months.	Death.	Clay, Journal of Anat. and Physiol., 1879, p. 498.	Lymphoid.	Thymus gland.
12	M.	Posterior mediastinum.	Side of chest: heart pushed to right; surrounded the vagus; left side of chest nearly full of liquid.	Pain in lower part of chest; cough, with traces of blood in sputa.	About 8 months.	Death.	Church, St. Bartholomew's Hosp. Reports, xiv., 1878.	Lymphoma.	
17	F.	Anterior mediastinum; reached from thymus to diaphragm, and laterally to each lung.	Attached to pericardium; lungs involved; glands at root of neck enlarged; glands all over body enlarged.	Dyspnœa; urine laden with lithates, but otherwise normal; temperature ranged from 96° to 104.2° F.	13 months.	Death.	Bennett, Intrathoracic Growths, London, 1872, p. 148.	Lymphadenoma.	

TABLE VI.—MISCELLANEOUS DISEASES OF THE MEDIASTINUM.

AGE.	SEX.	AREA INVOLVED.	OTHER PARTS AFFECTED.	CHIEF SYMPTOMS.	DURATION.	RESULT.	BY WHOM AND WHERE REPORTED.	VARIETY.	PRIMARY SEAT.
8 mos.	F.	Glands of posterior mediastinum.	Glands red and fleshy, but caseous; enlarged thymus.	Crowing respiration.	Not stated.	Death.	Goodhart, Brit. Med. Jour., April 12, 1879, p. 542.	Enlarged glands.	Mediastinum.
8 mos.	M.	Glands of anterior mediastinum.	Thymus enlarged; pressed on sternum.	Dyspnœa.	Not stated.	Death.	Goodhart, Brit. Med. Jour., April 12, 1879, p. 542.	Enlarged glands.	
22 mos.	Not stated.	Posterior mediastinum.	Ulcerated into mediastinum; bodies of second, third, and fourth cervical vertebræ carious.	Neck swollen; hoarseness; fetid breath, and cough.	3 months.	Death.	Journal Général de Méd., 1807, tom. xlii.	Foreign body.	Carious bone.
2½ years.	F.	Enlarged mediastinal glands; no tubercle.	One gland opened into trachea and caused death.	Dyspnœa and fits.	2 months (?).	Death.	Goodhart, Brit. Med. Jour., April 12, 1879, p. 542.	Enlarged glands.	Mediastinum.
4	Not stated.	Middle and posterior mediastinum.	Bronchial glands very large and tuberculous, involved pulmonary arteries, veins, and vagi.	Constant pain in epigastrium; rapid respiration; cyanosis.	7 weeks.	Death.	Gravenhorst, L'Union Méd., Feb. 5, 1867, p. 254.	Tuberculous glands.	
5 or 6	Not stated.	Middle mediastinum.	Opening five inches deep between œsophagus and trachea; this opening communicated with trachea.	Vomiting; rapid emaciation.	A few days.	Death.	Edin. Med. Jour., 1848.	Foreign body.	
7	M.	Posterior mediastinum chiefly.	Inflammation of œsophagus, of purulent character; pleurisy and peritonitis.	Ascites and hydrothorax; dyspnœa and pleurisy.	Not stated.	Death.	Eberth, Deutsch. Arch. f. Klin. Med., Bd. xxviii., Heft 1.	Mycotic mediastinitis.	Mediastinum. Scarlet fever (?).

Child.	F.	Glands in mediastinum.	Cavity in right lung; glands between bifurcation of trachea and superior vena cava were diseased.	Cyanosis; coma; œdema of face.	Not stated.	Death.	Baseri, Jahrb. f. Kinderkrankheiten, 1878, xii. 415.	Tuberculous glands.	
9	M.	Anterior mediastinum.	No post-mortem.	Not stated.	3 weeks.	Still alive.	Jones, Brit. Med. Jour., 1880, i. 286.	Cystic tumor.	Anterior mediastinum. Blow on chest.
Boy.	M.	Anterior mediastinum.	Pericardium contained pus.	Great thirst; pulsating tumor of sternum.	1½ months.	Death.	Rich and Bowen, Liverpool Med. and Chir. Jour., 1882, ii. 344.	Pulsating tumor of sternum.	
Child.	Not stated.	Entire mediastinum.	Emphysema of lung connecting with mediastinum.	Symptoms of emphysema.	....	Death.	Baerwinkle, Schmidt's Jahrbüch., vol. lxxxii. p. 63.	Emphysema.	
15	F.	Entire mediastinum.	Heart displaced by growth; aorta involved; also the pulmonary artery.	Pain; cough; rapid pulse.	2 months.	Death.	Goetz, Berlin. Klin. Wochenschr., 1885, xxii. 83.	Tumor; variety not stated.	
18	M.	Anterior mediastinum.	Tubercles in lung; hypertrophy and suppuration of thymus gland.	Pain in chest; oppression and cough.	....	Death.	Wittich, Arch. f. Path. Anat., tom. viii.	Thymus.	
10	M.	Anterior and posterior mediastinum.	Bronchial glands and glands in lumbar region enlarged; spleen, liver, and kidneys enlarged.	General dropsy; anæmia and weakness.	1 year.	Death.	Wilks, Trans. Path. Soc., Lond., vol. x. p. 259.	Lardaceous deposit.	"Glandular system."
11	F.	....	....	Diagnosis made by physical signs; cyanosis and pain in chest.	3 years.	Not dead when reported.	Rosenberg, Beiträge zur Casuistik der Mediastinal-Tumoren bei Kindern.	Not stated.	The report of this case is very incomplete and indefinite.

PART IV.

DISEASES OF THE CIRCULATORY, HÆMATOPOIETIC, AND GLANDULAR SYSTEMS.

FUNCTIONAL DISORDERS OF THE HEART.

By J. M. DA COSTA, M.D., LL.D.

FUNCTIONAL disorders of the heart occur in children from exactly the same causes as in adults. But they are not such common affections, because some of the causes are rarely or never present, as, for instance, sexual aberrations, hysteria, the abuse of tobacco, of alcohol, of coffee. Functional disorders of the heart in childhood are usually owing to gastro-intestinal irritations, to worms, to teething, or to anæmia, and in point of frequency those due to gastro-intestinal irritations stand first.

The *symptoms* presented by functional cardiac disorders of childhood are the same as in adults, especially palpitation, shortness of breath, irregular action, and uneasiness or pain. A child is, fortunately, oblivious or ignorant that it has a heart; hence the mental disquietude or distress, the fear engendered by watching the disturbed action, has no counterpart in the manifestations of the case. The palpitation, as in adults, comes on at uncertain times, and is sometimes distinctly provoked by exercise unsuited to tender years, as by walks too long. The shortness of breath, or, more strictly speaking, rapidity of breathing, bears a relation to the palpitation. But it is not excessive; and, as the respiration in infants and young children is normally far more variable than in adults, the fluctuations in the breathing are much more marked. The irregular action of the heart, its intermission, its perverted rhythm, its slow beats followed by beats hurrying to make up for the delay, are the most characteristic features of the functional cardiac disorders of childhood. Yet the degree of the disturbance has not the same value attached to it as in adults; for up to about the seventh year the heart's action is often of unequal strength and rhythm, prone to be irregular in the healthiest children during sleep, and greatly influenced by the acts of breathing. But when the irregularity is marked and persistent during the waking hours and during quiet breathing, it bespeaks a cardiac disorder, except in those instances in which, joined to other foreshadowings of a cerebral malady, it points to meningeal disease. Of the symptoms of functional affection of the heart mentioned, pain is the least conspicuous; indeed, it is much rarer than in adults; uneasy cardiac sensations, too, are seldom to be found.

The *physical signs* of the disturbed action of the heart in early life are in the main the same as later,—increased impulse, normal percussion-dulness, distinct second sound, and first sound either weak and short or sharp and valvular. The outlines of the percussion-dulness are difficult to determine with accuracy. Functional murmurs and those of blood-origin are, in my experience, of great infrequency.

It is needless to go into any details as to the *diagnosis;* it is apparent from what has been said of the symptoms and signs, if to these we add the evidences of the malady which has secondarily disturbed the heart, especially of the most common causes, gastro-intestinal disorders or anæmia. The latter state will betray itself not simply in the appearance, but also in headache, fretfulness, and sleeplessness. When the disturbance is lithæmic, as it occasionally though not often is, the condition is generally inherited. Besides the history, pain in the joints, and deposits of urates in the urine, give then a clue to the irregular cardiac action.

There is yet another form of irregular action of the heart, which is of all the most peculiar, and has not received the attention it deserves,—a form in which the irregular rhythm seems to constitute the whole malady; at all events, there are neither anæmia nor lithæmia, nor gastric nor intestinal disorders, nor worms, nor is, indeed, anything to be found except the irregular heart. These cases may be called idiopathic. I have seen them both in boys and in girls, in boys more frequently than in girls, and the children are often ruddy, and appear typically healthy except in their circulation. The heart's action is at times preternaturally slow,—in the sixties, or even the fifties. Intermissions are common, or a series of small beats followed by fuller strokes is noticed. The first sound is apt to be somewhat defective; the organ is always very impressionable. It exhibits in the most marked manner the influence of the respiratory acts, especially in deep breathing, and becomes very irregular if the breath be held. Medicines depress the heart quickly. I have known quinine in moderate doses send the pulse to forty-four, without rendering the rhythm more regular. On the other hand, during any febrile state the first effect—and, indeed, a lasting effect until the temperature declines—is the disappearance of the irregularity in the accelerated pulse. The age at which the changed rhythm shows itself is from three to six years: it is very rarely found in infants. It may be intensified by the disturbance of dentition, but it neither appears nor disappears with dentition. I have encountered the affection in children with impressionable nervous systems; in one case there was considerable twitching of the muscles of the face, yet it continued when this stopped. It is, indeed, more common in excitable children; but I have also met with it in those of stolid temperament.

It seems to run in families. Thus, I have watched two brothers, children of a mother with an extremely slow heart, who both are typical instances of the malady under consideration. In the eldest, now twenty-one years of age, the irregularity was first distinctly noticed at the age of six. He has had an excellent digestion; teething did not specially influence the pulse, which has always been rather accelerated, and, from his sixth year on, never free from intermittency; occasionally, too, it presents halting rather than arrested beats. During the first stimulus of any febrile attack the pulse invariably becomes regular. The irregularity has lessened much in the last years, but it has not disappeared. The pulse is eighty-four when quiet, the temperature normal; the respirations show nothing peculiar; the intermissions are very distinct after strong breathing. While at college, he became aware that he could row with force for a time only, and could not run long, yet he could swim for a mile. The brother, two years younger, has similar symptoms, but the changed rhythm shows itself especially in extreme slowness.

Neither of these kinds of cases nor of any one of the whole functional group is the *pathology* more certain than it is in the functional cardiac maladies of adults. There may be histological and chemical changes, but they have not been detected. The affection may have its starting-point in a weak muscle, or, what is vastly more probable, in the nervous system. The suddenness with which it often appears, and its disappearance without traces, are greatly in favor of this view. But the exact seat of the nervous disturbance it is not always possible to make out. In some cases the disorder of the cardiac nerves is clearly reflex; in others, the irritation points to the cervical portion of the spinal cord, or to the cervical sympathetic. Few instances only indicate disorder of the cerebral centres. In some, it is conceivable that the nervous mechanism within the heart itself is at fault. The idiopathic functional disorder, the impressionable heart described, may be of this character; though it is possible, and indeed more probable, to explain it by localized disturbance of the centres in the medulla, or, more likely, of the cardiac centre in the cortex of the brain. These matters must for the present, however, remain matters of pure speculation.

The *prognosis* of the functional disorder is a favorable one: the cause being removed, the malady ceases. This is certainly true of the affection as seen in consequence of gastro-intestinal maladies, and of anæmia. It is also true, though the process is a slower one, in children who have inherited or acquired lithæmia, and in the disturbed heart after fevers or from malaria. The most tedious and least promising cases are those of the idiopathic disorder or "impressionable heart." They last for years. I have watched cases from early childhood to young adolescence, as in the cases of the college students referred to, where the irregularity still exists; on the other hand, I have seen it gradually disappear after puberty, both in boys and in girls, but not immediately. The most important question connected with the whole matter is, whether in this or in any other form of the functional affection organic disease ever follows. I am not aware of any records on the subject, and can, therefore, only say, from personal experience, that I have not met with a single instance. Dilatation of the heart would be the condition most likely to occur; but I have never seen it happen. A certain amount of readily-disturbed action and of breathlessness on exertion is, however, apt to remain. Yet I have known a boy who had for years presented a marked instance of the impressionable heart become a champion runner,—it is true, only for dashes. Another interesting question also here claims solution. As there is so strong a nervous element in many instances of the cardiac disorder, is this the forerunner of chorea, epilepsy, or other kinds of neurotic ailment? Appealing again to individual experience, I have never traced any such connection. In what appear like associate cases the nervous malady is the first to show itself.

In the *treatment* of the functional cardiac disorder of childhood, it is evident that we must chiefly aim at removing the cause of the affection. A number of cases will be found to yield without much difficulty to careful attention to diet and to correcting digestive disorders. It is especially important that large amounts of food should not be taken at one time. An occasional laxative, too, will be beneficial, and a strict watch for worms will suggest whether vermifuges should be employed. The lithæmic state will require the same directions, especially as to diet, as gout or lithæmia in adults. The anæmic heart is benefited by a liberal meat diet, by iron, or by small doses of arsenic long continued, and by attention to life in the open air and to sufficient sleep. Moderate exercise suits all cases, and I have even seen benefit from making the little patients run certain distances daily, carefully graduated to suit their strength and slowly increased. Light gymnastics are also serviceable, steady, graded day by day, stopped when tiredness comes on, and not limited to exercises for the arms, but made to include movements for the legs. Nor need out-door sports, if not abused, be interdicted, except it be rowing. From sea-bathing, provided the bath be not too long and the skin be well rubbed afterwards, I have seen the happiest effects. Thus, in a young girl with the idiopathic irregular heart above described, whose case I watched for years, a cure was brought about by three weeks' steady sea-bathing. She had taken digitalis off and on for many months, always with temporary benefit, but not with permanent result; the pulse, after the cessation of the treatment, returned to upward of ninety, and intermitted from every sixth to every fourteenth beat. Her general health was excellent, and she lived a good deal in the country amid the best surroundings. It was a disappointment that the establishment of menstruation did not make a change in her cardiac condition. After the course of sea-bathing, the irregularity ceased, there remained for a time a little hurrying of the heart subsequent to a number of beats, and now, a year afterwards, the heart is always steady and not above eighty.

Greater stress has been laid, in the management of the functional disorder, upon remedies which remove the cause and upon hygienic means than upon so-called heart tonics. Indeed, these, I think, ought, as a rule, to be used only for temporary purposes. When they are called for, digitalis will be found to be the most trustworthy among them.

Should it be decided to give it with a view to its more sustained action, it is best administered in courses of about a month each, with an interval of ten days between each course. This treatment it is my habit to direct to be carried out for four to six months, and then only to resume it from time to time as may be necessary. The preparation usually employed is the tincture, prescribed in some pleasant vehicle, shortly after meals; the dose is from three to five drops for a child six years of age. In many instances a morning and an evening dose are sufficient, and after the heart becomes more regular a single evening dose will keep it so. This dose may be stopped at about the end of the fourth week of treatment, when generally the influence of the remedy is quite perceptible and the pulse for the time being steady. But it does not at once so remain, and further courses will be required. Whether in resuming the medicine we are to give more than the evening dose, depends upon the character of the effect observed; it is generally necessary to do so, at least in the second course.

Belladonna is also a drug of use in the functional disorder, either as a temporary substitute for digitalis or in combination with it. From chloride of barium, too, good results may be obtained, and in children old enough to take it in pills, containing about one-twentieth of a grain, it is both an excellent and a convenient remedy.

CONGENITAL AFFECTIONS OF THE HEART.

By WILLIAM OSLER, M.D., F.R.C.P.

THE deviations from the normal which occur in the heart during fœtal life result from (1) interruption to the natural course of development of the organ, (2) endocarditis, and (3) a combination of both these processes.

Nothing is more difficult in the consideration of these congenital affections than to assign, in special cases, the part played by each of these two important factors; indeed, it is often impossible. Various classifications have been adopted, none of which can be considered entirely satisfactory; perhaps as useful as any would be division into—

I. Conditions in which structures normal to the fœtus persist during extra-uterine life, such as open foramen ovale, persistency of the Eustachian valve, and patency of the ductus arteriosus.

II. True anomalies of development, as absence or imperfection of the ventricular septum, absence of the auricular septum, anomalous division of the truncus arteriosus, transposition of the great vessels, and numerical variations in the valve-segments.

III. Conditions caused wholly or in part by endocarditis, as extreme stenosis of the cardiac orifices, puckering, thickening, and adhesion of the valve-segments.

Here seems to be the most appropriate place for a few general remarks on the subject of *fœtal endocarditis.*

Practically there is but one form of inflammation of the endocardium met with in the fœtus,—that which corresponds in the adult to the chronic or sclerotic variety. Warty or verrucose endocarditis rarely occurs. A case is reported by Ayrolles[1] of a child, healthy at birth, which died on the tenth day slightly cyanosed. At the post-mortem the mitral orifice was extremely narrow, the right heart greatly enlarged, and the segments of its valves presented numerous vegetations covered with fibrin.

Certain structures occur on the valves which are often confounded with endocardial vegetations. Albini[2] described on the auriculo-ventricular valves of the new-born small nodular bodies consisting of translucent connective tissue which subsequently became firm and opaque. Bernays (of St. Louis) in his able paper on the development of the heart-valves states that these nodules of Albini represent the remains of fœtal structures. There may be ten or fifteen of these bodies, more commonly six or eight.

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[1] Revue mensuelle des Maladies de l'Enfance, 1885.

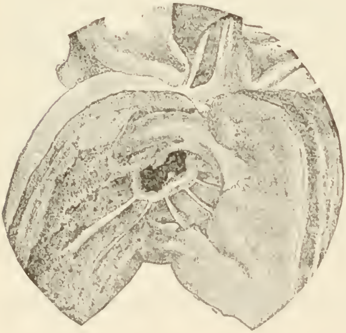
[2] Wochenblatt der K. K. Gesellschaft der Aerzte in Wien, 1856.

On many occasions I have had to correct a post-mortem diagnosis of endocarditis based on the existence of these nodules; and there can be no doubt that the extraordinary frequency of endocarditis described by Bouchut and by Labadie-Lagrave (nine-tenths of all cases dying of febrile affections) receives here its proper explanation.

It is not uncommon in the heart-valves of children to meet with small, rounded, bead-like bodies, usually of a deep purple color, which have frequently been mistaken for endocardial outgrowths. In reality they are small hemorrhages, and when the blood has undergone changes they certainly look not unlike little vegetations which have become smooth and round. They usually occur in healthy valves, but I have seen them several times upon segments which have presented sclerotic congenital changes.

Such a condition as is represented in Fig. 1 gives a characteristic picture of ordinary fœtal endocarditis. The segments are thickened, particularly at the edges, shrunken, and smooth. In the case of the auriculo-ventricular valves the cusps become united, and the attached chordæ tendineæ are thickened and shortened. In the semilunar valves all trace of the segments usually disappears, leaving a stiff, membranous diaphragm perforated by an oval or rounded orifice. As already stated, it is often difficult to say whether such a condition has resulted solely from a fœtal endocarditis or whether it is not a developmental error. In the majority of cases the truth would seem to be that in the anomalous valve chronic endocardial processes have occurred, leading to puckering and causing additional deformity. In many of the anomalies usually regarded as produced by fœtal endocarditis we would have to suppose the process beginning in the embryo during the second month, in a structure the entire length of which is not more than fifteen to twenty millimetres,—a supposition which is scarcely conceivable.

FIG. 1.



The results of fœtal endocarditis are most commonly seen in the right heart. This would appear to be not so much on account of the greater degree of liability *per se*, as because the valves of this side most often present errors of development.

Rauchfuss, whose experience at the Foundling Asylum at St. Petersburg has been exceptionally large, has met with the condition almost equally at the aortic and at the pulmonary orifice. In cases of stenosis and of obliteration, with perfect ventricular septum, he concludes that fœtal endocarditis is only more common in the right heart when in association with errors of development, whereas apart from such the left heart is not less frequently involved.

In persons who have reached maturity these abnormally-developed sclerotic valve-segments are especially liable to be attacked, and we find in the literature reports of many cases in which, associated with defects evidently of a congenital nature, there has been verrucose or even ulcerative endocarditis. The destructive processes may lead to perforation of the septum, or they may occur upon the thickened margin of an open foramen ovale or upon that of the defect in the ventricular septum.

To the general practitioner congenital heart-disease has an extremely limited interest. A great majority of the cases are still-born, or do not survive many weeks or months.[1] Instances which reach maturity are extremely rare. I do not call to mind more than one or two cases, in a somewhat extended hospital practice, of subjects of congenital heart-disease in persons who had passed the age of puberty.

The main scope of the present article is to give a concise account of the chief congenital cardiac affections, referring the reader for details to Peacock's work[2] and to the exhaustive monograph of Rauchfuss.[3] It will be most convenient to take up in order the anomalies of position, then those of the various portions of the heart and vessels, without attempting to follow any special classification.

A. GENERAL ANOMALIES.

1. *Acardia.*—Absence of the heart is met with in the monstrosity known as acardia, and need not be further referred to here.

2. *Double Heart.*—Double heart has been met with occasionally in the lower animals, usually associated with such extreme grades of deformity as trichocephalus.

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[1] A large amount of valuable material of this sort goes to waste annually. If practitioners would take the trouble to send to the Army Medical Museum (which can be done free of charge) the bodies of infants suspected to be the subjects of cardiac disease, particularly such anomalies as anencephalic monsters, umbilical hernia, and spina bifida,—forms which are particularly liable to be associated with cardiac anomalies,—the profession would within a few years have at Washington a collection of cardiac anomalies which would be of the greatest value for reference.

[2] Malformations of the Heart, 2d ed., London, 1866.

[3] Gerhardt's Handbuch der Kinderkrankheiten, Bd. iv., Abth. 1.

Lancereaux[1] refers to a case described by Collomb in which an opocephalous monster possessed two hearts.

3. *Bifid Apex.*—Externally the heart rarely presents any special changes except in association with the conditions, hereafter to be described, in which there are but two or three cavities. Occasionally the apex of the heart is bifid. In a heart which I obtained a short time ago there was a fissure an inch and a half long at the apex and on the anterior wall. Specimen 7796 of the Army Medical Museum, Washington, is the best of the kind which I have seen.

4. *Dextrocardia.*—Transposition of the heart is met with either as a part of a general transposition of the viscera, or, in rare instances, alone. It has a purely anatomical interest. The condition is sometimes termed intrathoracic ectocardia of the lateral variety.

5. *Mesocardia.*—Mesocardia is a condition in which the organ occupies a central position in the chest-wall, such as is normal at the earliest period of development.

6. *Ectopia Cordis.*—This condition is associated with fission of the anterior chest-wall, and usually also with that of the abdomen. In its most extreme grade the organ may lie free, or it may be immediately beneath the skin behind a congenital sternal fissure. Three varieties are usually described,—the cervical, the pectoral, and the abdominal. In the first, the most rare, the heart is situated in the neck, and may be in contact with the tongue, or even with the palate. It is always associated with other extensive anomalies. The second form usually exists with marked fission of the thoracic wall. The organ may be entirely free; more commonly it is covered with the pericardium, or with this layer and the skin. In the third variety, abdominal ectocardia, the organ lies below the diaphragm, in the upper part of the abdomen. In one remarkable case the organ occupied the position of the left kidney. Ectopia cordis is rarely compatible with extra-uterine life, except in cases of the abdominal variety, with which persons have lived for many years.

7. *Absence of Pericardium.*—Here may be mentioned the rare anomaly absence of the pericardium, which is often found in association with ectocardia, but also as a separate anomaly in a heart otherwise normal. The defect may be only partial.

B. ANOMALIES OF THE CARDIAC SEPTA.

I. TOTAL DEFECT OF THE SEPTA.

Total defect of the septa of the auricles and of the ventricles is rare. When present the condition of cor biloculare, or, as it is sometimes called, "reptilian" heart, exists. An exceptionally good example of this rare anomaly is described by Dr. William P. Northrup.[1] The child was a "blue baby," which lived one month. As shown in Fig. 2, there were only two cavities. The venous chamber had the outline externally of the two auricles with their appendices; within there was no attempt at partition. The cavæ were normal. No pulmonary veins entered the auricle. The ventricle showed within a ridge,—the rudimentary septum. The pulmonary artery was represented by a fibrous string. There was no trace of any orifice, and its trunk was impervious as far as the ductus arteriosus. This vessel was pervious and equal in size to the innominate artery, and it gave off branches to the lungs. The aorta was given off from the middle line of the ventricle, and was normal.

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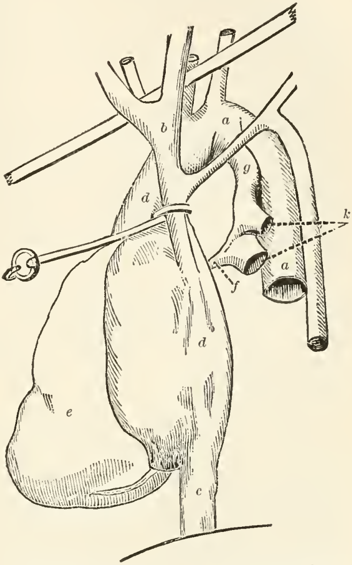
[1] Traité d'Anatomie pathologique, tome ii.

Fig. 2.



Life Size.—*a*, aorta; *b*, vena cava superior; *c*, vena cava inferior; *d*, *e*, auricle and ventricle; *f*, rudimentary pulmonary artery; *g*, patent ductus arteriosus; *k*, vena azygos anomalous.

II. ANOMALIES OF THE SEPTUM ATRIORUM.

These are of frequent occurrence: some of them are unimportant; others lead to serious disturbance of the circulation.

(*a*) Perforations in the form of small canals between the membranous portion covering the foramen ovale and the muscular septum are by no means uncommon, and are without clinical significance.

(*b*) Still more common is it to find an oblique opening beneath the membrane of the fossa ovale, the margin of which at one point has failed to become attached to the annulus. Often the valvular slit is large enough to admit the handle of a scalpel, and in cases of great dilatation of the auricle there may be a lozenge-shaped orifice, owing to the stretching of the septum. Sometimes the valvular communication is double, or there may be three narrow slits.

(*c*) The membrane of the foramen ovale may itself present several perforations, or it may exist as a cribriform structure. In an extreme grade of this condition there may be only a few fibrous filaments crossing the orifice.

(*d*) Permanent patency of the foramen ovale is the condition of the septum which has excited the greatest attention, and scores of cases are on record of persons, of all ages, dying of various diseases, in whose septum auricularum no trace existed of the normal membrane of the foramen ovale.

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[1] Proceedings of the New York Pathological Society for 1888.

Two groups of these cases may be recognized,—first, those in which the anomaly existed with other serious defects, such as narrowing of the pulmonary artery or of the conus arteriosus, or defect of the ventricular septum,—conditions which produce abnormally high pressure in the right auricle.

In the second group the patent foramen ovale has been the only condition present, and there may not have been symptoms indicative of cardiac trouble. Such cases have been met with accidentally in persons dying of various diseases, without record of cardiac distress. Sometimes (as in the case reported by Henry I. Bowditch,[1] of a woman, aged forty-five, who had been troubled from her nineteenth year) there may be occasional attacks of lividity and dyspnœa on exertion. The margin of the orifice may be thickened by a chronic endocarditis. I have twice seen (once at the University College Hospital in a patient of Sir William Jenner's, and the other a specimen in Germany) extensive recent endocardial vegetations on the edges of the orifice, similar to those mentioned in the case of Claudius Amyand.[2]

This condition is a congenital defect,—simply a failure in the development of the membrana fossæ ovalis. I doubt its occurrence as the result of atrophy. In cases of enormous distention of the auricle it is not uncommon to see the membranous septum greatly stretched, even to the extent of producing a pouch-like saccular distention; yet I do not remember ever having seen an opening caused in this way.

(*e*) Rarer, but more serious, are the extensive defects of the muscular portion of the auricular septum, in which case the opening is in the anterior portion of the auricle,—not posteriorly, as in defects of the membranous septum. Here also the sickle-shaped margin looks towards the posterior part, while in the latter it is directed anteriorly. It occasionally happens that there is extensive defect of both the muscular and the membranous portions of the septum. It may be so extensive as to leave only a single undivided cavity. This, however, rarely occurs without other serious anomalies of development.

(*f*) *Premature Closure of the Foramen Ovale.*—Premature closure is occasionally met with, in which at term the orifice is practically closed, the auricles communicating only by a narrow valvular slit. I do not know of any instances in which there has been at birth total occlusion: a valvular slit, however narrow, constantly remains. The cases are not very common: I met with it in a child born at term with general anasarca. The heart was large, and the right auricle was distended. The foramen ovale looked as if completely closed, but on passing a probe around the margin of the fossa it entered a valvular orifice eight millimetres in length; the heart was otherwise normal. The ductus arteriosus was very large,—almost equal to the aorta in size. It was sixteen millimetres in circumference, and the aorta was seventeen millimetres. The figure annexed shows this great enlargement of the ductus arteriosus. Such a condition of the ductus would indicate, it seems to me, that the foramen had been virtually closed for some time, and that the blood from the inferior cava had followed the course of the adult circulation, increasing the work of the right heart and gradually leading to the enlargement of the duct. The connection of this with the dropsy of the fœtus is not very clear. As Peacock was able to collect only three cases of premature closing of the foramen ovale, the condition must be extremely rare.

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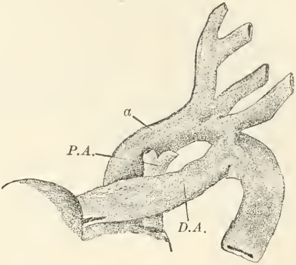
[1] Boston Medical and Surgical Journal, 1861, vol. lxiv.

[2] Philosophical Transactions, 1747.

Fig. 3.



Great Enlargement of the Ductus Arteriosus in a Case of Premature Closure of Foramen Ovale.—*a*, aorta; *P.A.*, pulmonary artery; *D.A.*, ductus arteriosus.

III. DEFECTS OF THE VENTRICULAR SEPTUM.

Defect in the ventricular septum is an extremely common condition. It may exist alone, though it is more commonly associated with lesions of the orifices. Total defect of the septum is not common. When existing, and the septum of the auricles is at the same time absent, the cor biloculare, or so-called reptilian heart, exists; whereas when the septum of the auricles is present, and that of the ventricles absent, the condition is that of cor triloculare.

Rokitansky, from whose monograph[1] on this subject the greater part of our information is derived, divides the septum ventriculorum into a posterior part lying between the auriculo-ventricular orifices and an anterior part lying between the two arterial orifices; while between these is the membranous portion,—the pars membranacea septi, the so-called undefended space of English authors. Until the appearance of Rokitansky's work this membranous portion had been regarded as the part most commonly absent in septum defect; but he showed that it is the anterior portion which stretches between the pars membranacea and the anterior wall that is most often defective. The posterior part of the ventricular septum may be partially or completely absent; it is usually associated with defect of the membranous portion, and also of the septum atriorum. It is not nearly so common as absence of the anterior portion of the septum. When complete, which is not usual in this form, there is a large orifice, and the conus arteriosus of the right side is quite rudimentary. Defect of the anterior portion of the septum is more frequent where it lies just beneath the arterial orifices and is in the muscular substance. Most common of all, as Rokitansky has shown, is defect of the hinder section of the anterior septum lying just anterior to the membranous portion. Rokitansky throws some doubt upon the existence of congenital defect of the membranous portion of the septum, but there appears very little doubt that it does occasionally occur, though the others referred to are much more frequent.

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[1] Die Defecte der Scheidewand des Herzens, Wien, 1875.

C. LESIONS AND ANOMALIES OF THE VALVES AND ORIFICES.

I. ANOMALIES OF THE SEMILUNAR VALVES.

Small defects in the lunated spaces of the semilunar valves occur so frequently that they can scarcely be called anomalies: even when extensive they do not cause symptoms.

The only anomalies of importance are the reduction and increase in the number of the valve-segments.

(*a*) *The Bicuspid Condition of the Sigmoid Valves.*—We do not yet fully know the steps in the development of the semilunar valves, but it occasionally happens, after division of the truncus arteriosus, that in the folds which bud out to form the segments some disturbance occurs which results in the formation at the aorta and at the pulmonary artery of two instead of three valves. That this in many cases is truly a congenital anomaly, and not the result of endocarditis, pre- or post-natal, is evident from the occurrence of instances in which the fused segments look quite normal,—smooth and clear, and free from any traces of endocarditis. Such a specimen as is here figured could certainly not have resulted from any affection of the valves. Of twenty-one instances of this anomaly of which I have notes, two were found in the fœtus at term.[1] I have met with it most frequently in the aortic valves. In twenty-one instances only two occurred in the pulmonary valves. On the other hand, Dilg[2] was able to collect from the literature sixty-four cases in the pulmonary artery, and only twenty-three in the aorta. Viti[3] has found the anomaly most frequent at the aortic orifice in the proportion of seven to three. Between a condition in which the margins of two segments are not united directly upon the aortic wall, but are joined on a sort of raphé which holds them some distance from it,—between this and conditions in which the two segments seem to have perfectly fused, forming a single valve, with perhaps only a slight trace of the division into the two sinuses of Valsalva, all grades of the anomaly are met with. A specimen in the Army Medical Museum at

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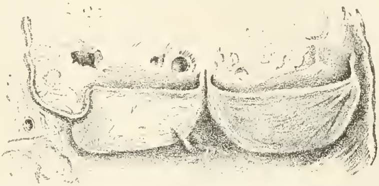
[1] Transactions of the Association of American Physicians, vol. ii.

[2] Virchow's Archiv, Bd. xci.

[3] Lo Sperimentale, 1886.

Washington is one of the most perfect which I have seen; the measurements of the two valves are practically equal,—five centimetres each,—and it is difficult to say which is the combined segment, as there is scarcely a trace of any indication of division on the arterial side. There has been some discussion as to the possible origin of this condition in a fœtal endocarditis. Virchow has maintained this view for a large proportion of the cases.[1] The occurrence of the anomaly in the fœtus without a trace of endocarditis shows that in certain of them, at any rate, there is an error in development. Peacock inclines to this view, as do also Sperino and Martinotti[2] and Martinotti[3] in their valuable memoirs on this subject.

FIG. 4.



CONGENITAL FUSION OF TWO SEGMENTS OF THE AORTIC VALVES, WITH SECONDARY SCLEROTIC THICKENING.—Specimen from a man aged twenty-one years.

It is usually an easy matter to distinguish between congenital cases and those in which, as the result of chronic sclerotic endocarditis, the partition between the two segments has been destroyed. In the former case the two valves approach each other very nearly in size, and in the latter the single valve bears the usual proportion to the others. When in the aorta this anomaly is not usually associated with any other cardiac defect, but when in the pulmonary artery (as shown by Dilg's table) defect of the septum ventriculorum is extremely common,—in fifty-six of the sixty-four cases. I was much struck with the fact that in all my cases, up to the time of the report above referred to, the valves which were fused were the segments behind which the coronary arteries were given off, and I suggested that in some circumstances associated with the development of the coronary arteries the explanation of the anomaly might be found; but in two of three specimens which I have seen in the past two years the coronary segments were not alone involved.

Clinically this is a most important congenital valvular defect,—not in itself, as it is probable that the two segments can close the orifice, and I doubt if in the primitive condition regurgitation occurs. The danger results from the extreme liability of the abnormal structure to undergo sclerotic change. So common is this that of the many cases which I have seen only the fœtal ones did not show signs of thickening and deformity. In fifteen of the cases which I have reported death resulted directly or indirectly from the lesion. The average age at time of death in the cases of aortic defect was very much higher than in the pulmonary artery cases, probably because the latter is so often associated with other serious anomalies.

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[1] Virchow's Archiv, Bd. ciii. S. 103.

[2] Sulle Anomalie numeriche delle Valvole semilunari aortiche e polmonari, Torino, 1884.

[3] Le Anomalie numeriche delle Valvole semilunari del Cuore, Torino, 1886.

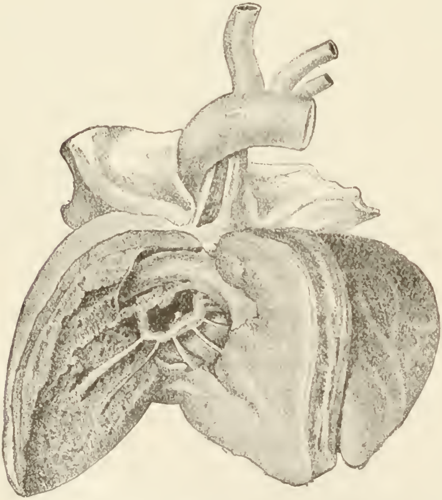
(*b*) *Increase in the Number of Valves.*—Supernumerary valves are not very uncommon; they occur more frequently in the pulmonary artery than in the aorta. Usually the number is increased by a small additional segment. Dilg (*loc. cit.*) has collected reports of twenty-four cases of four pulmonary and two cases of four aortic semilunar valves. I have met with two instances, one in a fœtus and one in an adult, both of the pulmonary segments. One of the four valves was a smaller segment than normal, and its free edge was on a slightly lower level than the contiguous valves. The sinus Valsalvæ was well marked in each case, but the corpus Arantii was not present. This would appear to be the rule. Though the four valves may be of equal size, the supernumerary segment is, as a rule, smaller, and is apt to be fenestrated. This anomaly is not so likely as the preceding one to be accompanied by other defects.

Five semilunar valves have been met with in a few instances. Dilg has collected three cases, one at the aorta and two at the pulmonary orifice.

II. OF THE AURICULO-VENTRICULAR VALVES.

Changes in the auriculo-ventricular valves are the result of anomalous development or of fœtal endocarditis. In the tricuspid valve there may be imperfect separation of the cusps, so that there is only a membranous diaphragm with a large circular orifice. The valve may show four distinct cusps. In certain instances of transposition of the vessels the bicuspid valve has been found on the right side. Anomalies of a similar kind are occasionally met with at the mitral valve, the segments of which may be imperfectly differentiated or increased in number to three.

Fig. 5.



Stenosis of the Tricuspid Orifice, with Thickening and Adhesion of Valve-Segments.—Child aged four months.

Endocardial changes in the tricuspid are the most common, and usually coexist with affections of the pulmonary valve or with serious congenital defects. Stenosis from adhesion or thickening of the segment is a most common condition, and is beautifully illustrated in Fig. 5. It is very rare to find definite vegetations.

Atresia of the orifice may result from a developmental anomaly in which there may be no appearance of any valvular mechanism, a condition invariably associated with other profound disturbances. Obliteration of the orifice by fœtal endocarditis is more common, and can be recognized by the marked changes in the endocardium. Sometimes, however, it is a difficult matter to determine which factor has prevailed.

III. LESIONS AND DEFECTS AT THE PULMONARY ORIFICE.

These practically may be considered under three headings: (*a*) stenosis of the orifice, (*b*) atresia of the orifice and of the artery, and (*c*) stenosis of the conus arteriosus.

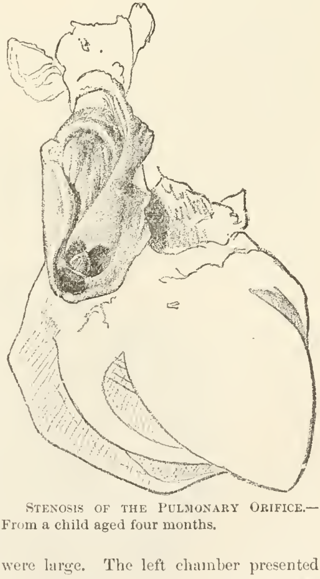
(*a*) *Stenosis of the Pulmonary Orifice.*—This forms one of the commonest and at the same time one of the most important of the congenital cardiac affections, resulting in the majority of cases from fœtal endocarditis. The following case, from my Montreal records, is a typical illustration of this condition:

A child aged four months, well nourished, and of average size. From birth it was noticed that the complexion was rather leaden, but he throve like any other healthy infant. During a slight attack of bronchitis the cyanosis deepened, and he died after a few days' illness.

The heart, which is shown in Figs. 5 and 6, was greatly hypertrophied, and the right auricle was enormously distended: a small-sized billiard-ball could be fitted into the chamber. The foramen ovale was not quite closed, presenting a small narrow slit. The tricuspid orifice was small; the segments of the valves were contracted and thickened, the edges red and swollen. On the posterior segment there was a small colorless pedunculated vegetation. The chordæ tendineæ were much thickened and shortened.

The right ventricle was enormously hypertrophied, the wall measuring from ten to twenty-seven millimetres in thickness. The conus arteriosus was narrowed, measuring only seventeen millimetres in circumference. Near the ring the pulmonary orifice was greatly narrowed, admitting with difficulty a probe less than one millimetre in diameter. The valve-segments had united, and, as shown in Fig. 7, had left a narrow slit-like orifice, the edges of which were very firm and hard, but without vegetations. The sinuses of Valsalva were large. The left chamber presented nothing abnormal. The ventricular septum was perfect, and the ductus arteriosus was closed.

FIG. 6.



STENOSIS OF THE PULMONARY ORIFICE.—From a child aged four months.

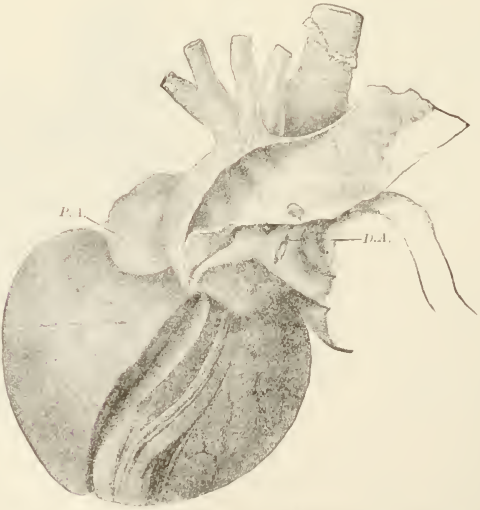
This typical example illustrates the condition usually found. The narrowing results from a slow endocarditis, which gradually causes adhesions of the segments and contraction of the orifice, without, as a rule, any signs of active changes. From the smooth membranous condition of the valve in certain cases, it is difficult to resist the belief that the stenosis may also be the result of faulty development. Subsequent to birth a recent warty endocarditis may be grafted upon the old sclerotic segments, and in many of the published cases the orifice, or even the pulmonary artery, has been described as blocked with vegetations.

Case 608 of the post-mortem records of the Montreal General Hospital illustrates this point. Girl, aged eighteen; death with symptoms of chronic valve-disease. The right ventricle was greatly hypertrophied. The pulmonary orifice was stenosed; only two millimetres in diameter; the valve-segments thickened and adherent. There were numerous vegetations extending from the orifice to the tricuspid valve, and at the base of these there was an abscess-cavity in the wall of the heart. There were vegetations also in the tricuspid segments.

Cases of this group offer closer analogy to the lesions of adult life than any other form of congenital heart-disease. The hypertrophy of the right ventricle, which reaches a very high grade, as is shown in Fig. 5, may for years compensate perfectly the valvular defect, particularly if the septum of the ventricles is imperfect.

(*b*) *Atresia of the Pulmonary Orifice.*—Entire obliteration of the orifice of the first part of the pulmonary artery is tolerably common in congenital heart-disease, though less frequently met with than stenosis, and is prob-

Fig. 7.



Atresia of the Pulmonary Artery, *P.A.*, and Persistence of the Ductus Arteriosus, *D.A.*

ably always the result of a developmental defect. It is a more serious condition than the one just described, and is of necessity associated with other anomalies, such as imperfection of the ventricular septum and persistence of the ductus arteriosus. The following is an excellent illustration, from my Montreal records:

A. B., a well-developed male infant, aged thirteen days, cyanosed from birth. The child had many paroxsyms of dyspnœa, and died in convulsions. The heart was large, its circumference at the base being twelve centimetres. The right auricle was large, and the foramen ovale only partially closed. An oval orifice, five by three millimetres, communicated with the left chamber. The tricuspid orifice was large, and on the auricular face of the segments were numerous gelatinous vegetations. The right ventricle was greatly hypertrophied. The conus arteriosus was narrowed to a short funnel-shaped tube which ended in a *cul-de-sac*, corresponding to which, on the exterior of the heart, was attached a narrow, cord-like vessel representing the pulmonary artery. It passed as a narrow tube for seven millimetres, and widened gradually until it reached a point where the ductus arteriosus joined the main branches. The ventricular septum was imperfect in its upper part, the orifice measuring nine by seven millimetres. The lower border was formed by the muscular walls of the septum. The endocardium upon it was thickened, and there were fresh endocardial beads. The upper part of the orifice was bounded by a thin translucent membrane, upon which there was a mass of beaded, gelatinous vegetations. The left chamber presented nothing abnormal. From the aortic arch the large ductus arteriosus passed to the pulmonary artery at its bifurcation.

(*c*) *Stenosis of the Conus Arteriosus.*—This remarkable anomaly forms a considerable portion of the cases of obstruction at the pulmonary orifice. Assmus,[1] in his extensive article upon the subject, has collected forty-seven instances. It consists essentially of a narrowing of the infundibulum or conus of the right ventricle. The grade of the stenosis may be extreme, so that the orifice may admit only a small probe. It is probably always the result of developmental disturbance, not of mural endocarditis. Subsequently, as is so often the case, there may be inflammatory thickening. The septum of the ventricle is always imperfect; the foramen ovale may be open, and the ductus arteriosus patent.

These three lesions of the pulmonary orifice, taken together, constitute for the practitioner the most important of all the congenital cardiac disorders. Of one hundred and eighty-one instances, collected by Peacock, of various congenital malformations, one hundred and nineteen cases came under this category.

The duration of life is considerably higher than in any other form: according to Assmus, twenty-three per cent. died in the first year of life, twenty-six per cent. survived the twelfth year, and sixteen per cent. the twentieth year; one woman reached the fifty-seventh year. Peacock states that of forty-five cases of congenital heart-disease which lasts beyond the twelfth year thirty-eight, or eighty-six per cent., are the subject of pulmonary-orifice disease, so that in a case which has survived the fifteenth year the probability of the existence of this form of defect is extremely great. This is particularly true if the ventricular septum is imperfect.

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[1] Deutsches Archiv f. Klin. Medicin, Bd. xx.

Rokitansky states that three months is the longest period to which he has known life to be prolonged when the stenosis was unaccompanied by imperfection of the septum. In the case above reported the duration of life was a little longer than this.

IV. LESIONS AND DEFECTS AT THE AORTIC ORIFICE.

These are the same in kind, although not so frequently met with, as those of the pulmonary orifice. They are due partly to developmental errors, the result of abnormal division of the common truncus arteriosus, or, in exceptional instances, the result of fœtal endocarditis. There may be stenosis or atresia of the orifice, or narrowing of the left conus arteriosus. Rauchfuss has collected twenty-four cases of stenosis and atresia with perfect ventricular septum, in the majority of which there had been endocardial processes. Cases of atresia at this orifice are much more numerous in proportion than at the pulmonary orifice. Stenosis and atresia may also exist with defect of the septum, in which case it most probably results from anomalous fission of the truncus arteriosus.

Stenosis of the left conus arteriosus is rare. Dilg (*loc. cit.*) has collected fifteen cases, of which seven appear to have had their origin in inflammatory conditions, whereas in eight there was evidence of defective formation in the conus arteriosus. The duration of life in aortic-orifice disease is not nearly so great as in the pulmonary-artery affections. In thirty-three cases of atresia and stenosis, with or without defect in the septum, only one survived the first month of life. On the other hand, it is curious to note that of the sixteen cases of stenosis of the left conus arteriosus tabulated by Dilg the majority were in adults. His own case was a child nearly two years of age; the rest of the cases were all over eighteen and ten of them over thirty years of age.

D. LESIONS AND ANOMALIES OF THE LARGER VESSELS.

I. TRANSPOSITION OF THE AORTA AND THE PULMONARY ARTERY.

This somewhat rare anomaly is most frequently met with in association with other congenital defects, such as umbilical hernia, spina bifida, hydrocephalus, and talipes, but it may occur with or without other serious cardiac changes.

The following illustrative case is from my Montreal records:

A fœtus of the eighth month presented all the malformations above referred to. The heart was not much enlarged; the right auricle was of moderate size; the cavæ were normal; the Eustachian valve was large, and the foramen ovale open. The tricuspid valves presented two bead-like hemorrhagic nodules. From the right ventricle, which was larger than the left, a vessel was given off, eight millimetres in width at the root, which passed over a vessel emerging from the left ventricle, crossed the left bronchus, and then descended as the thoracic aorta. Seven millimetres from its origin it gave off a small pulmonary branch to the imperfectly-developed lungs, and, just before it reached the spine, the left subclavian passed off vertically to the first rib. The left ventricle was smaller than the right. The mitral orifice and valves were normal. From this chamber a vessel passed up on the trachea without communicating with the vessel from the right ventricle; it then divided into the innominate and left carotid arteries. In the septum ventriculorum was a small orifice, the size of a goose-quill, at the upper and back part of the septum. The semilunar valves in both vessels were abnormal; there were only two on the branch from the left ventricle, and in that one from the right two of full size and between them a tiny imperfect one.

Rauchfuss has collected twenty-five cases of this kind of anomaly. In some instances the lungs were supplied from the vessels of the right chamber, which, as in the case just described, subsequently formed the descending aorta. But in the cases of perfect transposition the pulmonary artery arises from the left ventricle. The duration of life in this anomaly is, as a rule, short: twenty of the cases did not survive the first year.

II. PERSISTENCY OF THE DUCTUS ARTERIOSUS.

Premature involution of the ductus arteriosus occurs occasionally during fœtal life. It has been met with in extreme narrowing of the pulmonary arteries.

Normally this vessel gradually undergoes obliteration, and by the fourteenth day is permanently closed. Nothing could be added to the full description of this process in the first volume of this work by Dr. J. Collins Warren. Interruption of this normal involution is not very uncommon, usually occurring with other anomalies, rarely alone. It is most frequently met with in connection with stenosis or atresia of the pulmonary artery or narrowing of the isthmus of the aorta. The vessel forms a short, wide, funnel-shaped canal, the distal end being the larger. Hypertrophy of the right ventricle and dilatation of the pulmonary artery occur in a majority of the cases, and in those which live for some years atheromatous processes are extremely common in the vessel. In the analytical table given by Morrison[1] of forty-six cases of patency of this vessel, the pulmonary artery was closed in thirty-four and open in eleven, the foramen ovale was closed in seven and open in thirty-four, and the foramen ovale and ventricular septum were open in twenty-seven. Of sixteen cases seven died in childhood, from three months to nine years. Of the remainder five reached ages from nineteen to thirty-four, and four lived to between forty and fifty. (Rauchfuss.)

In a few cases patency of the ductus arteriosus has existed without other anomalies in persons who have reached maturity. In one case there was no cyanosis; in another, only a very slight grade. The ductus arteriosus may be absent,—an extremely rare condition.

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[1] Practitioner, February and March, 1888.

III. HYPOPLASIA OF THE HEART AND AORTA.

A condition of smallness of the heart and of the great vessels was referred to by many of the older writers, but it was not until Virchow in 1856 called special attention to it in connection with chlorosis that the subject aroused the general attention of pathologists. More recently the elaborate measurements of Beneke have given us accurate ideas of the relative size of the vessels at different periods of life. He states that the collective lumina of the arteries are relatively narrow, in proportion to the body-length, up to the age of puberty, but that at this time the arteries rapidly enlarge, and the heart acquires a great increase in its power, undergoing what he calls its puberal development. He regards a congenital smallness of the heart as a cause not only of general feebleness, but also of retardation or disturbance in the development of this period. It is quite possible, as has been suggested, that many of the cases of palpitation of the heart in young persons from sixteen to twenty years of age, particularly in association with nervousness and anæmia, may be due to overstrain of the heart not equal to the demands of a rapidly-growing body, and to a too quick expansion of the arterial system.

Upon this condition the section in Rauchfuss's monograph, as well as the work of Beneke,[1] is worthy of the most careful study.

SYMPTOMS OF CONGENITAL HEART-DISEASE.

The symptoms fall naturally under two divisions, general and local.

General Symptoms.—*Cyanosis.*—Over ninety per cent. of the cases present, in a greater or less degree, lividity of skin, indicating the circulation of imperfectly-aerated blood in the superficial capillaries. So distinctive is this symptom of congenital cardiac defect that the term "morbus cæruleus" has been applied to it, and "the blue disease" is a synonyme for congenital heart-affection.

The admirable study recently made by Dr. Alexander Morrison, in which he has carefully analyzed seventy-five cases of congenital heart-disease, has given us full information on the frequency of this symptom, and on the special conditions with which it is commonly associated. His figures are of sufficient importance to be given in some detail.

The ductus arteriosus was open in forty-six cases, closed in eighteen, doubtful in eleven. In these forty-six cases the pulmonary orifice was open in eleven and closed in thirty-four; the foramen ovale was open in thirty-four and closed in seven; the interventricular septum was deficient in thirty-five and perfect in eleven ; the foramen ovale and the interventricular septum were deficient in twenty-seven ; the foramen ovale was closed and the interventricular septum was perfect in four.

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[1] Die anatomischen Grundlagen des Constitutionsanomalien des Menschen, Marburg, 1878.

Of the forty-six cases cyanosis existed in a minor degree in nineteen, in a more pronounced degree in thirteen, was absent in four, and doubtful in ten.

Of the eighteen cases in which the ductus arteriosus was closed, the pulmonary artery was open in fourteen and closed in four ; the foramen ovale was open in twelve and closed in five ; the ventricular septum was open in thirteen and closed in five ; the foramen ovale and septum ventriculum were open in seven and closed in none. Of these eighteen cases cyanosis was absent in two.

The following are among the more important facts which he states must be taken into account in considering the mechanism of cyanosis in cases of cardiac malformation. In a considerable majority of cases—seventy-one per cent.—the ductus arteriosus is patent. Of these only twenty-three per cent. had an open pulmonary orifice, as compared with seventy-seven per cent. in the group with the ductus arteriosus closed,—a ratio which is nearly reversed as regards closure of the pulmonary orifice. "As regards the relative significance of special lesions in cyanosis, closure of the pulmonary orifice and patency of the foramen ovale and the ventricular septum are most frequently associated with it, and, conversely, the patency of the pulmonary orifice and the closure of the foramen ovale and the ventricular septum are associated with relative absence or a minor degree of that state."

A discussion of the various theories which have been brought forward to explain this symptom does not come within the range of a practical work of this kind. It is sufficient to say that the two which have been most widely accepted have been that of Morgagni—who referred it to the general congestion of the venous system due to obstructive processes similar in kind, though less in degree, to those which occur in various affections of the adult—and that of William Hunter, who attributed the discoloration to the admixture of the venous and arterial blood. The former view was maintained by Louis and by Moreton Stillé, whose inaugural thesis at the University of Pennsylvania in 1844 was a masterly summary in favor of Morgagni's theory.

The congestive view is now very generally accepted, and the work of Dr. Morrison seems to make it additionally clear that "the main though not the only factor in the production of cyanosis is the inadequate aid afforded to the circulation by diminished lung-functions." So far as I know, chronic emphysema is the only condition, other than congenital heart-disease, in which we see patients cyanosed for days or even longer and yet able to get about or even to walk to hospital. Here certainly it is a matter of diminished lung-function.

The cyanosis in the majority of cases of cardiac anomalies appears early, within the first week of life ; it may gradually disappear, to recur under conditions of excitement or upon exertion. The external temperature is reduced, owing to the prolonged stay of the blood in the superficial capillary vessels, and the patient often complains of chilliness. The internal temperature of the body is apparently not reduced. Dyspnœa, particularly on slight exertion, and cough are common symptoms. The child does not thrive as do other children: it is usually feeble and dwarfed, and often displays a lethargy both of mind and of body.

Clubbing of the fingers and of the toes is a very characteristic feature in cyanosis of congenital heart-disease.

Local Symptoms.—The patient may not complain of any cardiac distress, and a physical examination may be necessary to determine the nature of the trouble. Inspection may show marked bulging of the præcordia, more particularly in the sternal region and over the third, fourth, and fifth left costal cartilages. This is usually associated with strong heaving impulse in the lower sternum and even in the epigastrium.

Palpation may discover a thrill which is more common towards the base than at the apex of the heart.

Percussion-dulness is, as a rule, increased, particularly towards the right, and, as Gerhardt has remarked in cases of pulmonary-valve disease, along the left margin of the sternum even as high as the second rib.

Auscultation does not always reveal the presence of a murmur, but in the majority of instances there is a loud bruit, systolic in time, with the maximum intensity towards the base of the heart. Diastolic murmurs are much less commonly heard.

DIAGNOSIS.

The diagnosis of patency of the ductus arteriosus has been made in several instances,—more often, indeed, than it has been confirmed post mortem.

Cyanosis has been very frequently recorded. In the forty-six cases analyzed by Morrison it was absent in four, present in a minor degree in nineteen and in a more pronounced degree in thirteen, and doubtful in ten. The physical signs are chiefly those of great hypertrophy of the heart, particularly of the right ventricle. Gerhardt has referred to the marked prominence in the upper cardiac region, and to the extension of the area of dulness to the left of the sternum, reaching as high as the second rib, due to the dilated and hypertrophied conus arteriosus and the distended pulmonary artery. A loud systolic murmur in the second and third left interspaces and in some instances a diastolic murmur have been present which may completely obliterate the sounds. There are no unequivocal physical signs.

The diagnosis of congenital narrowing or closure of the pulmonary orifice has a greater interest than that relating to any one of the congenital defects, owing to the fact that in these cases the duration of life may be prolonged for many years. Cyanosis and its accompanying phenomena are usually present; the pulse is small, often irregular; palpitation is complained of; there may be cardiac pain. Physical examination shows more or less prominence of the præcordia, such as almost invariably accompanies hypertrophy of the heart in children. Percussion shows enlargement of the right heart, the dulness extending beyond the right border of the sternum, and the maximum impulse is frequently in the region of the xiphoid cartilage. There are instances, indeed, in which the impulse has here been marked, although scarcely detectable in the normal situation. A thrill, systolic in time, not propagated into the carotid, and of maximum intensity towards the right base, is common. On auscultation there is a systolic murmur of maximum intensity over the conus and pulmonary orifice in the region of the second and third left costal cartilages. The second pulmonary sound is usually feebler than the aortic, and in some instances has been accompanied by a diastolic murmur.

Atresia of the pulmonary orifice with open ductus arteriosus may also be accompanied by a systolic murmur of maximum intensity at the pulmonary cartilage. The first sound may be reduplicated. The rhythm may be disturbed. It may be extremely irregular, or, in some instances, the shortening of the diastolic pause gives a fœtal tic-tac character to the sounds.

Although the clinical diagnosis of this condition has been correctly made in many cases, yet it must be confessed that errors are very common, and cases of defect of the septum and persistency of the ductus arteriosus and congenital tricuspid stenosis cannot always be distinguished from pulmonary-valve disease. The statement of Peacock, above referred to, as to the enormous percentage of affections of this orifice in persons cyanosed from birth who have survived the twelfth year, is an important collateral factor in the diagnosis of such cases.

The following instances illustrate some points in the diagnosis of these cases:

Charles W., aged twenty-two months, seen with Dr. J. Madison Taylor May 5, 1887. He was the sixth child. Nothing special was noticed until the ninth day, when the mother observed that he was blue, particularly when he cried. He throve well until the seventh month, when he lost power on the right side, particularly in the arm, which was still affected at the time of observation; the movement of the fingers was particularly imperfect. The child looked healthy; the head was well formed, though the anterior fontanel opening was one and one-fourth inches. The general color of the face was good, though the lips were blue. The finger-tips were livid, the nails quite cyanotic. The dorsal veins of the fingers were remarkably distended, rather more on the left than on the right side. There was a wavy pulsation in the vessels of the neck. The cardiac impulse was seen just below and within the nipple-line. On palpation it was to be felt just outside the nipple-line, but was not specially forcible; there was no thrill. The dulness extended from the upper border of the fourth rib; to the right it did not extend beyond the sternal border. On auscultation at the apex there was a loud shock, with a ringing first sound, and a soft, distant, systolic murmur. As the sternum was approached this became louder, and it had a maximum intensity on the third rib and in the second left interspace. It was well heard also outside this interspace, gradually disappearing towards the axilla. At the angle of the scapula both sounds were heard free from murmur. At the aortic cartilage the sounds were clear,—the second ringing.

James D., aged ten years, idiot, inmate of the Pennsylvania Institution for Feeble-Minded Children. He was feeble as a child, and did not walk until his fifth year; he is now small and not sufficiently developed for a child of his age. There is intense cyanosis, particularly of the face and of the hands; the finger-tips are quite livid; the terminal phalanges are clubbed, as are the toes also; the tongue protrudes, and has, with the lips, a distinct bluish tinge. The chest is large, much flattened, and depressed in the lower axillary regions. The costal margin is strongly everted and the sternum rostrated. The heart-impulse is seen in the epigastrium, not in the nipple-region. The pulse is small and feeble.

On palpation the impulse is not forcible. The shock of sounds can be felt in the nipple-region. There is no thrill. The area of absolute cardiac dulness is diminished.

On auscultation a loud systolic murmur in the nipple-region obliterates the first sound, is propagated into the axilla, and is heard at the scapula. Towards the sternum it increases in intensity, and the maximum is over the fourth left costal cartilage; here the murmur is very intense, but both sounds are heard. In the area of pulsation in the epigastrium the murmur is also heard, but the sounds are here of an entirely different character, owing to the echo which accompanies them. There is here a distant but very distinct diastolic rumble. At the aortic cartilage the systolic murmur is feeble; the second sound is louder at the aortic than at the pulmonary cartilage. The lungs are everywhere hyper-resonant. The breath-sounds are loud and breezy, and expiration is a little prolonged.

These cases illustrate the difficulties encountered in arriving at a satisfactory diagnosis of the exact lesions. In the first case, the child twenty-two months old, the position of the maximum intensity of the murmur over the third rib and in the second left interspace was the only really definite physical sign. Whether it was caused by stenosis of the pulmonary artery, imperfection of the auricular septum, or patency of the ductus arteriosus, could not, I think, be determined.

In the second case the loudness of the murmur at the apex and its transmission to the scapula, just as in mitral disease in the adult, suggest involvement of this orifice; on the other hand, the maximum intensity of the murmur on the sternum and at the fourth left costal cartilage, and the occurrence of a diastolic rumble not present at the apex, are very suggestive of tricuspid disease, and the case is very possibly one of involvement of this orifice with imperfection of the septum.

The majority of all cases of congenital cardiac defect do not survive birth more than a few hours or a few days. In stenosis of the pulmonary artery there are cases on record in which the patient has lived to a comparatively advanced age; and, as has already been mentioned, the same holds good in instances of narrowing of the conus arteriosus of the left ventricle. In cases of open foramen ovale with patency of the ductus arteriosus, adult life has been reached. Death commonly results from affections of the lungs, sometimes from hæmoptysis, and very often from tuberculous disease. Dropsy rarely supervenes.

TREATMENT.

The treatment of congenital heart-disease is largely hygienic. Fresh air, avoidance of cold and of all conditions liable to induce bronchial irritation, and a carefully-regulated diet constitute the most essential elements.

In attacks of dyspnœa the child naturally assumes the position in which it can most effectually inspire,—usually a sitting posture, with the shoulders more or less fixed. Bloodletting, under these circumstances, might be freely employed, as is done with good effect in cardiac dilatation and in emphysema.

As to medicinal agents, the peroxide of hydrogen in eight-minim doses three times a day has been recommended by Sir William Foster. Brisk saline cathartics are also very beneficial. Digitalis must be used with care, but when hypertrophy begins to fail and the dilatation to increase we have nothing to take its place. In the distressing dyspnœa, which is often very worrying at night, Hoffmann's anodyne, chloroform with spirit of camphor, and whiskey are of service. Hypodermic injections of ether will prove of value in cases in which the use of digitalis is contra-indicated.

ENDOCARDITIS, ACUTE AND SUBACUTE.

By W. B. CHEADLE, M.D., F.R.C.P.

Definition.—Inflammation of the lining membrane of the heart, affecting chiefly the fibrous structure of the cardiac valves and their tendinous attachments.

Etiology.—Endocarditis is probably never idiopathic or primary except in case of direct injury, but always secondary to some other affection. It occurs most commonly in connection with acute articular rheumatism, but it arises also in the course of chorea and of the specific fevers, especially scarlet fever, less frequently in measles and erysipelas, and more rarely still in enteric fever and variola. It occurs likewise as a complication of puerperal fever and other forms of septicæmia and pyæmia, and occasionally during pregnancy or after parturition, and as a result of syphilis.

The influence of chorea in producing endocarditis has been referred to attrition or disorder supposed to arise during the valvular movements. Yet, as the cardiac force is usually much lessened in chorea, it is not easy to see how any condition of more forcible friction can exist. The connection with chorea is probably solely through the rheumatic state. Recent observations as to the occurrence of subcutaneous nodules and other rheumatic manifestations in chorea, and especially in chorea in which heart-disease occurs, render it highly probable that the endocarditis of chorea is entirely of rheumatic origin. It is possible that in some cases a soft mitral murmur is produced by atony or paresis of the cardiac muscle and consequent leakage of the valve from imperfect closure, as suggested by Dr. Sturges;[1] but in the majority the lesion is organic: if the murmur subsides, it comes back later and remains. Dr. Wilks holds that all mitral systolic murmurs associated with chorea are organic; Dr. Sansom has come to the same conclusion. Dr. Stephen Mackenzie[2] found evidence of permanent heart-disease absolutely certain in sixty per cent., and possibly in over eighty per cent., of cases of choreic murmur examined from one to five years after. I have no exact statistics upon this point, but all my observations point very strongly to the conclusion that the mitral and aortic mur-

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[1] Chorea, p. 56.

[2] Trans. Internat. Med. Cong., 1881, vol. iv. p. 104.

murs of chorea are almost invariably organic: first, because of the frequent association of rheumatic arthritis, chorea, and endocarditis; secondly, because I have so frequently seen serious valvular disease eventually develop after chorea, when there was at the time only a soft passing murmur, or reduplication of the second sound, or even no alteration of the heart's sound at all during the primary chorea; thirdly, because of the frequent occurrence of organic heart-disease in which the chief pathological antecedent is chorea; fourthly, the significant association with pericarditis. The choreic valvular affection, being then organic, must be ascribed to endocarditis. Endocarditis does undoubtedly take place frequently in chorea. The morbid appearances met with in the valves in organic heart-disease connected with chorea are exactly those produced by endocarditis. In the recent cases there are the beadings and deposit of fibrin. In those of older standing there are the same thickening and puckering and contraction which are seen to follow endocarditis from other causes. Endocarditis being, then, clearly at any rate the chief cause of choreic heart-disease, the question arises as to the nature and cause of endocardial inflammation. The close association of chorea with rheumatism in a large proportion of cases,[1] on the one hand, and the close association of endocarditis with rheumatism on the other, naturally suggest that the endocarditis is rheumatic. Pathologically it is impossible to distinguish the morbid appearances and results of an endocarditis associated with rheumatism from the same condition associated with chorea; and constantly we find chorea, endocarditis, and articular rheumatism together.

It is a very significant fact, moreover, that endocarditis picks out especially the cases of chorea associated with rheumatism. Out of eighty-four cases of chorea of which I have accurate notes with regard to these precise points, in sixty-two there was a history of rheumatism in the patient or in near blood-relations, and in the remaining twenty-two no history of rheumatic taint. In the sixty-two rheumatic choreas there was organic heart-disease in forty-three, or 69.3 per cent.; in the twenty-two cases in which no rheumatism could be traced there was organic heart-disease in six only, or 27.2 per cent. The statistics of the Collective Investigation Committee[2] show a similar discrepancy, although it is less marked,—viz., fifty per cent. as against thirty-five per cent. This is owing partly perhaps to less complete and minute inquiry by a large body of busy practitioners of varying degrees of observing power and thoroughness, and partly to the fact that they take into account only antecedent and concurrent arthritis as evidence, omitting other manifestations of rheumatism. There is, then, a special connection between the endocarditis of chorea and the endocarditis of rheumatism, and it is highly probable that whenever it occurs in relation to chorea it is of rheumatic origin, even if there be no positive history of other manifestation of the disease.

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[1] The Collective Investigation Statistics of the British Medical Association, vol. iii., give from thirty-nine to forty-four per cent. of antecedent or immediately associated rheumatic arthritis. Dr. Barlow estimates the proportion of proved connection at fifty-seven per cent. My own statistics give fifty-five per cent. Dr. Sturges places it at about twenty per cent.; but, as this relates only to previous well-marked attacks of acute rheumatic arthritis, it is clearly inadequate. See previous article on Rheumatism, vol. i. p. 803.

[2] Rep. of Coll. Inv. Comm. of Brit. Assoc., 1887, vol. iii.

Endocarditis, again, which sometimes arises in relation to erythema and to fibrous nodules is almost certainly rheumatic. As with chorea, the special connection of these with rheumatism renders their appearance in a case of endocarditis strong presumptive evidence of its rheumatic nature.

Taking endocarditis in children, then, generally, it may be affirmed that it is dependent upon the rheumatic state in the vast majority of cases. Dr. West[1] gives statistics of one hundred and forty cases, of which in 62.1 per cent. rheumatism was either known or asserted on good grounds to have been the starting-point of the mischief. And this takes into account only pre-existing or concurrent arthritis, omitting all the large number of cases in which the rheumatic arthritis occurred later, together with those in which the evidence of rheumatism consists of other manifestations than arthritis, such as erythema and fibrous nodules. Dr. West (*op. cit.*) also quotes the estimate of M. Roger that seventy-eight per cent. of cases of heart-disease in children are rheumatic, and that of M. Cadet de Gassicourt, which places the proportion at eighty-two per cent. Dr. Goodhart, in two hundred and forty-eight cases of heart-disease, noted one hundred and fifty-four as rheumatic, or sixty-two per cent.; and this, like most of these statistics, includes only cases of antecedent or concurrent rheumatic arthritis or with rheumatic family history, and therefore is below the mark.

In a special investigation which I carried out for some years with regard to this point, of one hundred and five cases of which I have accurate record I find a clear history of acute rheumatic arthritis in the patients or near blood-relations in eighty-five, or eighty per cent. If the existence of chorea or other rheumatic phases were to be admitted as evidence, the proportion would be higher still.

It may be noted, further, that the other conditions in relation to which endocarditis is met with have also some close association with acute rheumatism, as, for instance, scarlet fever, in the course of which symptoms of acute rheumatism not unfrequently occur, also pregnancy and parturition, which are known predisposing causes of acute rheumatism, and chorea, likewise especially associated with the rheumatic state.

The fact of the association of endocarditis with conditions such as rheumatism, the specific fevers, pyæmia and septicæmia, erysipelas, and Bright's disease, is very suggestive of its immediate dependence upon the presence in the blood of some morbid material which by its irritant properties or by causing capillary stasis or thrombosis acts as the exciting cause of inflammation. When endocarditis occurs in the course of scarlet fever, it comes on usually in the stage of desquamation. In twelve cases of this kind recorded by Dr. West[1] it was accompanied by fever and anasarca, so that the association was possibly with the uræmic rather than with the scarlatinal poison, although the condition of the urine is not stated. But cases are recorded in which endocarditis supervened in the first few days of scarlet fever, when there was no sign of any renal affection. In many of these cases there were concomitant pain and tenderness of joints not to be distinguished from those of acute rheumatism. In Dr. Ashby's[3] cases referred to in the article on rheumatism the symptoms set in with great regularity about the end of the first week. In most of these slight cardiac bruits developed, but were not regarded as indicating endocarditis, and were not persistent. The condition was attributed to septicæmia rather than to rheumatism.

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[1] Diseases of Infancy and Childhood, 7th ed., p. 553.

Mechanical injury has been mentioned as a cause of endocarditis. This occurs chiefly in the case of a ruptured valve, probably through the impinging of the torn portion against adjacent membrane, causing friction or slight contusion with each successive contraction of the heart.

The existence of old-standing valvular disease is certainly a powerful predisposing cause of endocarditis. Possibly its action is mechanical in the same way, through the knocking or rubbing of excrescences against one another or against the lining of the valves, and possibly because in all tissues an inflammation once excited is renewed there with abnormal readiness.

Increase of tension is credited with being concerned in the production of endocarditis, this being founded upon the almost absolute limitation of endocardial inflammation to the left cavities of the heart; and it is pointed out that the position is reversed in the case of the fœtus, endocarditis being limited in that case to the right heart. It is clear that this is the situation of greatest tension in each case,—there being after birth greatest resistance and greatest propulsive power to overcome it, and therefore greater shock and friction, in the left heart, while before birth these are greatest in the right heart, owing to the undeveloped state of the pulmonary circulation. There is, however, also less difference between the blood of the left and right heart in the fœtus in utero, and it is possible that the rheumatic or other virus acquires greater potency in oxygenated arterial than in venous blood. The fact of the prevalence of chronic endocarditis and endarteritis in conditions such as Bright's disease gives some support to the tension theory; but it is at least just as likely that the effete matters in the blood may act as direct irritants, and this, and not increased tension, be the efficient cause of inflammation. Yet it must not be overlooked that the valves, where tension and friction are greatest, are the special seats of endocarditis, and tension may be regarded as one factor in its production, the other chief agent being a morbid condition of the blood.

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[1] Diseases of Infancy and Childhood, 7th ed., p. 554; see also the article on rheumatism in this work, vol. i. p. 788.

[2] Brit. Med. Jour., Sept. 15, 1883, pp. 514, 515.

Age has a distinct influence as a predisposing cause of endocarditis, probably chiefly through the rheumatic connection. Children are especially liable to it. It is impossible to agree with Rosenstein's assertion that the disposition to endocardial affections is not so great in childhood as after puberty. It is opposed to the experience of others. The results of the Collective Investigation Committee[1] give seventy-two per cent. in cases of rheumatism in children, as compared with about forty-six per cent. in adult males, although in the case of females the discrepancy is much less. It appears to be especially common between the ages of four and twelve. Under four it is certainly less common than after, yet is by no means unknown. Many cases are on record of the occurrence of mitral endocarditis in children a few months old.

The disease has been shown to occur even before birth. Mr. Bland Sutton relates a case of recent endocarditis in a fœtus of eight months. There was puckering and thickening of the mitral valve, and the margins of the aortic and pulmonary valves were fringed with soft vegetations. Other cases are on record of adhesion and thickening of the valves, probably caused by intra-uterine endocarditis.

Sex.—It does not appear that in the case of children sex exercises any marked influence upon the production of endocarditis: if a boy and a girl have rheumatism, their chances of endocarditis appear to be equal. But, seeing that more girls have rheumatism than boys,—and if we regard chorea as generally rheumatic the proportion is further raised,—more girls have endocarditis; and this probably throws light upon the singular fact that mitral stenosis is so common among young women: it is constantly a legacy of the rheumatism of childhood, in which mitral stenosis is so frequent a lesion.

In endocarditis from other causes there is probably an equality between the sexes; but on this point statistics are wanting.

Pathology and Morbid Anatomy.—The endocardium is a highly vascular membrane, and the valves equally so with the rest; capillary vessels are numerous, and vessels of some size are met with immediately beneath in the subendocardial connective tissue:[2] the conditions are thus favorable to the genesis of the inflammatory state.

There is another condition, I imagine, present in the case of children, which probably plays an important part,—viz., a readiness of tissues to proliferate, which is a characteristic of the period of growth. This is seen perhaps in the general tendency to connective-tissue growths during early life and in the extreme rapidity with which the heart hypertrophies in obstructive valvular disease. It also appears again in most significant relation to

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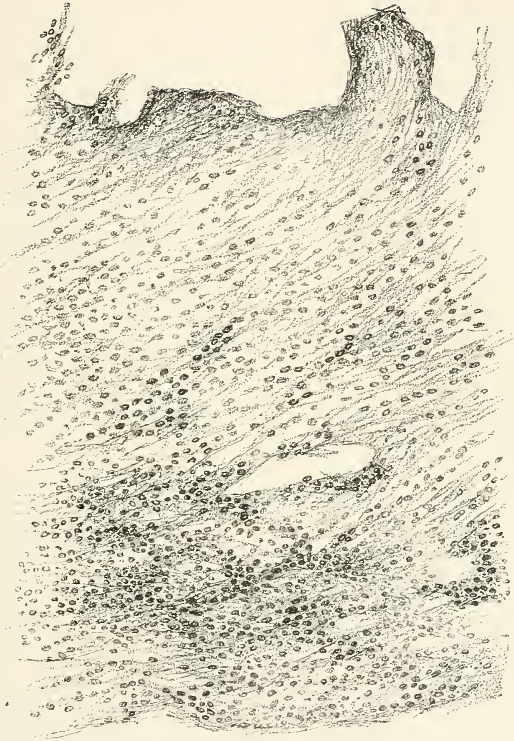
[1] Brit. Med. Jour., Feb. 25, 1888, p. 391.

[2] Klein and Noble Smith, Atlas of Histology, p. 148.



Section of Mitral Valve in a Case of Rheumatic Endocarditis in a Child.—Showing proliferation and cell-infiltration of sub-endothelial fibrous tissue. (Dr. Barlow, Hospital for Sick Children, Great Ormond Street. Ellen M., aged eight and a half years. March 28, 1888. Arthritis, nodules, morbus cordis, purpura.

endocarditis in the often-mentioned connective-tissue formation of subcutaneous nodules. It is a reasonable supposition, I think, that under similar stimulation the tissues of young subjects will proliferate more readily than those of the old. It has been pointed out, in speaking of the etiology, that the left side of the heart is chiefly implicated after birth. In certain cases the tricuspid valve is also the seat of endocarditis; sometimes, but not often, it is more extensively affected than the mitral or the aortic; more rarely still it is involved alone. The pulmonary valves invariably escape endocarditis, the left side then being the principal seat of inflammatory changes. In children, as in adults, of the two valves on that side the mitral is more often affected than the aortic. Out of one hundred consecutive cases of heart-disease in children examined by me, the mitral was affected alone in eighty-seven. Of these, sixty were instances of regurgitation, six of stenosis, and twenty-one of both regurgitation and stenosis; two were cases of aortic obstruction, one of aortic regurgitation, and five of aortic and mitral disease together. One was a case of simple dilatation, and five were instances of congenital pulmonary stenosis. This special implication of the mitral is attributed by Dr. Sibson[1] to the fact that the flaps of the mitral valve press against each other when the valve is shut with much greater force. The inflammatory changes, further, are earliest and most intense on those portions most exposed to friction and pressure,—viz., on the auricular surface of the mitral and the ventricular surface of the aortic, just within the free margin in each case. The chordæ tendineæ are often affected, and sometimes the general lining of the left cavities is also involved. In severe cases the muscular fibres beneath are implicated, and there is exudation into the spaces between them.

The morbid changes which take place in the structure of the valves and their tendinous attachments in endocarditis are of two kinds, and they are exceptionally well seen in the case of children. The structure of the endocardium consists of a layer of flat endothelial cells forming the internal lining; these rest upon a layer of branching connective-tissue cells, and below again comes a coarser trabecular layer of fibro-elastic connective tissue, which is continuous with and merges into the subendocardial connective tissue. The valves are folds of the endocardium held together by fibrous tissue between. The prime change of all, the first step in the morbid process of endocarditis, and the most striking feature of it, is the proliferation of the fibrous connective tissue immediately beneath the endothelium, —the branched-cell layer. With this there is some proliferation of the endothelium, and there is infiltration of the whole structure of the valve with leucocytes. But the hyperplasia of the connective tissue is the chief feature, and it is this principally which gives rise to the swelling of the valves. There is, in addition to the active cell-change in the valve-structures themselves, a deposit of fibrin on the surface, either an exudation from the vessels of the part or a precipitation directly from the general blood-stream. In this way are produced the small nodulated projections which we call vegetations. At first they appear in children as rows of red, gelatinous-looking, translucent beads at the margins of the valves, resembling closely in appearance the normal corpora Arantii of the semilunar valves, but very numerous; then as fibrin is deposited they become rougher and more opaque and grow larger. The swelling and deposit may be reabsorbed, or may increase to such a degree as to form excrescences large enough to interfere mechanically with the passage of blood through the orifice. The chordæ tendineæ may be similarly affected with bead-like swellings or vegetations. Portions of the vegetations are liable to be detached, and form emboli lodging in arteries in different parts, or, if the healing process goes on, cicatricial thickening and contraction follow and cause various distortions of valves, which lead to regurgitation or stenosis. Acute or subacute endocarditis is indeed in the case of children the usual —perhaps the invariable—starting-point of chronic valvular disease of the heart.

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[1] Reynolds's System of Medicine, Eng. ed., vol. iv. p. 458.

The cell-proliferation of the fibrous connective tissue of the valves which forms the basis of the vegetations and is the chief feature of endocarditis is of extreme interest in connection with the similar formations in the subcutaneous fibrous tissue,—the nodules. Microscopically the process seems to be identical. Thin sections of these nodules which I have examined show them to be composed of proliferating fibrous tissue,—wavy elastic fibres, together with the spindle nucleated growth which marks the transition-stage from cells to fibres. They are said to be highly vascular. Sections of the nodular projections of the inflamed cardiac valves show similar wavy elastic fibres and proliferating nuclear growth of the connective tissue beneath the endothelium.

In view of the close connection shown by Drs. Barlow and Warner and by Dr. Money to exist between the evolution of these nodules and the occurrence of endocarditis and pericarditis, this similarity of the histological changes in the subcutaneous nodules and in the beadings on the cardiac valves renders it in the highest degree probable that they represent analogous morbid changes set up by the same cause. The tissue of each proliferates under the stimulus of the irritation of the rheumatic virus.

The changes which follow acute or subacute endocarditis are both grave and numerous. Fibrous contraction and thickening and puckering or ulceration or perforation of the valves and tendinous cords, leading to narrowing of the valvular openings, or causing imperfect closure and regurgitation; consequent changes in the cardiac chambers, such as dilatation and hypertrophy; simple dilatation, partial or general, from injury to the muscular tissues of the walls by accompanying myocarditis; sometimes embolisms from the detachment of fibrinous concretions on the valves or from thrombi in the cavities,—all these occur in the case of children, as with adults, and will be treated of under their proper headings.

Symptoms.—Simple endocarditis, acute or subacute, uncomplicated by pericarditis or myocarditis, may run its course without giving rise to any cardiac symptoms. Not only may there be no cardiac pain or palpitation, or dyspnœa or distress of any kind, but there may even be no valve-murmur. This is shown by the fact that a patient may go through an attack of rheumatic fever without sign of implication of the valves at the time, and yet serious valvular mischief develop subsequently which can be accounted for only by antecedent endocarditis. And this masked endocarditis is especially common in the case of children, in whom the inflammation is particularly prone to be insidious and subacute.

In such cases the only symptoms are those referable to the disease which underlies the endocarditis,—the rheumatism, the pyæmia, or the scarlatina,—and its existence is therefore a mere matter of inference and suspicion. As was pointed out in the article on rheumatism, in the rheumatic endocarditis of childhood, where the distinctive joint-symptoms are slight or absent, the occurrence of endocardial inflammation frequently passes unnoticed. Oftentimes the general illness is so slight and featureless and transient that the patient never comes into the doctor's hands at all, or, if he does, the heart is not examined, because there is nothing to suggest directly its implication. This occurrence of endocarditis in connection with rheumatism where all the leading features of that affection as it is seen in adults, such as arthritis, sweating, and pyrexia, are ill defined or absent, and without notable cardiac disturbance, is, indeed, one of the special features of subacute rheumatic endocarditis in early life.

It is of the utmost importance, then, in the case of children, that in every condition in which it is possible that endocarditis might arise the heart should be carefully examined from time to time. In articular rheumatism, however slight, in chorea, in erythema, in tonsillitis, in septic and pyæmic conditions, in nephritis, this precaution should always be taken. Even in the slight febrile attacks so common in children from many and often unexplained causes, this should be done. Such febrile attacks may be the only general sign of endocarditis, and auscultation may reveal an unexpected murmur; and thus rest and treatment may save from disaster.

Another special feature of rheumatic endocarditis in children is its tendency to relapse and recur. The inflammation subsides and revives again, with simultaneous relapses in other symptoms,—a slight return of pyrexia, a fresh return of arthritis in some of the joints, a new crop of nodules, or an eruption of erythema. Cardiac murmurs perhaps appear afresh or gain increased intensity, or a new morbid sound appears, such as a reduplication of the first or second sound, or a presystolic thrill or rumble, or a pericardial rub. In these cases of relapsing endocarditis, extremely rapid and excited action of the heart is sometimes a striking feature, and the pulsations may reach one hundred and forty or one hundred and sixty in the minute. Or there may be little change in the cardiac signs, but all the while progressive endocarditis, leading to greater and greater valvular mischief, or serious thickening and strangulating adhesions of the pericardium from accompanying pericarditis.

Although it is possible that endocarditis may go on for a time without furnishing any distinctive symptoms, there is usually some physical sign, in the shape of a change in the cardiac sounds, a prolongation of the systole at the mitral or aortic valve, or an actual bruit, or a reduplication of the first or second sound, to indicate its presence. These may be discovered on auscultation, although there may be no constitutional symptoms, such as rise of temperature or pulse- or respiration-rate, traceable to the endocarditis. Frequently the development of a murmur or reduplication is the only evidence of the existence of endocarditis. The most common murmur of all is the simple systolic mitral, indicating regurgitation; next to this is the rumbling bruit before the systole, indicative of mitral stenosis: in nearly one-fourth of the cases (twenty-one out of eighty-seven), according to my statistics, the systolic mitral and the presystolic exist together. In a very small proportion of cases the murmur is basic and systolic, signifying aortic obstruction; more rarely still, diastolic, indicating aortic regurgitation. Of these the mitral systolic murmur is usually, the presystolic mitral invariably, organic and a sign of endocarditis. The aortic systolic murmur is rarely hæmic or functional. The diastolic aortic is invariably organic and a certain evidence of endocarditis.

Another sign of the advent of endocardial inflammation especially common in rheumatic endocarditis in children is reduplication of the second sound, audible at the apex, but not audible at all at the base of the heart. A reduplication of the second sound at the base is a frequent phenomenon, and its existence is easily explained by the difference in time of closure of the aortic and pulmonary valves, due to the difference of resistance in the systemic and pulmonary arterial systems respectively. It is met with in Bright's disease on the one hand, and in pulmonary obstruction on the other. But the reduplication of the second sound at the apex is less easy to understand. It must depend upon the asynchronous falling open of the tricuspid and mitral valves as the ventricles relax and the auricles begin to contract, for there is no other cardiac operation which occurs at this moment of diastole which would be audible at the apex and not at the base. It cannot be due to asynchronism of the aortic and pulmonary valves, for that is audible at the base of the heart only, not at the apex. The want of synchronism between the mitral and tricuspid valves is probably due to the inflammatory thickening of the mitral, which is thus rendered more rigid and yields less readily to the first force brought to bear upon it than the pliant healthy tricuspid. When the ventricle relaxes at the end of systole, the normal tricuspid at once flaps back in response to its suction-force and the weight of blood filling the auricle, while the more rigid mitral does not move, perhaps, until the contraction of the auricle, which begins a little later, comes into play.

With this reduplication of the diastolic sound there is often a distinct murmur following the second portion of the double sound,—a diastolic murmur, soft and blowing in character. The only explanation of this seems to be that the mitral, already becoming stiffened and swollen by the cellular proliferation of its connective tissue, closes effectively when driven forcibly to by the ventricular contraction, but springs slightly open again, like an ill-fitting door, when the pressure is relaxed, and does not fall back completely and closely against the ventricular wall, thus narrowing the orifice and causing sonorous eddies in the incoming stream from the auricle. Be this as it may, however, the interesting clinical fact remains, which I have verified by numerous observations, that this reduplication of the second sound limited to the apex, either with or without the accompaniment of a diastolic bruit, is the first stage or sign of mitral stenosis. The whole morbid change of sound may disappear and the valve resume its normal state; but far more often it remains and gradually changes into the true presystolic rumble. Dr. Sansom[1] has come to much the same conclusion as to the significance of reduplication generally. His experience leads him to think that in these cases endocarditis is followed by stenosis rather than by regurgitation. But I should limit the statement to reduplication of the second sound at the apex, and make it more absolute as to the connection with stenosis of the mitral valve. This reduplication of the second sound at the apex and diastolic bruit are, then, among the most certain signs of rheumatic endocarditis.

Occasionally the first sound is reduplicated, and this may be audible at both apex and base; but the exact meaning of this want of unison between the ventricles is still uncertain. It is possibly due to the first effect of increased resistance in the pulmonary arterial system, caused by leakage or obstruction at the inflamed mitral valve.

One important accompaniment of rheumatic endocarditis, rare in adults, but common in children, and of high clinical value, is the evolution of subcutaneous fibrous nodules, which have been previously described. In treating of the pathology of the disease it was shown that there is a close correspondence between the structure of these nodules and that of the bead-like excrescences upon the edges of the cardiac valves which form the foundation of the vegetations in endocarditis. I believe that Dr. Barlow's[2] suggestion, that the changes would prove to be analogous, is correct. In this view the relation between their appearance and the development of endocarditis and pericarditis is significant. Out of twenty-seven cases investigated by Dr. Barlow and Dr. Warner,[3] "there was reason to believe that some morbid cardiac condition obtained in every one." In five cases examined post mortem, mitral disease was found in all, and pericarditis in four. Dr. Angel Money found these nodules in half the cases of rheumatism in which well-marked heart-disease occurred, and in one fatal case of pericarditis in which they were present a distinct formation of the kind was observed invading the heart's substance and extending from the pericardium inward; and Dr. Barlow, in one case of pericardial adhesion with simultaneous evolution of nodules, observed that these adhesions had a distinctly nodular character. In nearly half of Dr. Barlow's cases the cardiac disease was seriously progressive; valvular murmurs increased and dilatation developed in spite of treatment. During the last few months I have had four cases in which plentiful and persistent evolution of nodules, in almost continuously successive crops, has proceeded *pari passu* with progressive endocarditis and pericarditis to a fatal issue.

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[1] Lettsomian Lectures, p. 18.

[2] Trans. Med. Cong., 1881, vol. iv. p. 118.

[3] Brit. Med. Jour., Sept. 15, 1883, p. 511.

The eruption of subcutaneous fibrous nodules, then, in any case, whether of recognized rheumatic arthritis or chorea or erythema marginatum, or appearing alone, must be regarded not only as a sign of the existence of rheumatism in some form, but also as gravely suggestive of the coexistence of endocarditis, and that a similar change to that observed in the fibrous tissues beneath the skin may be proceeding unseen in the cardiac valves.

The following case illustrates this form of relapsing or progressive endocarditis:

J. T., a boy of seven, admitted to the Children's Hospital in Great Ormond Street, December 1, 1887, complaining of pain and stiffness of the joints, with slight swelling. The doctor who attended him said that he was suffering from low fever. The condition was not recognized as rheumatic. He had never had rheumatism before, but had had two attacks of chorea, and his mother had had rheumatic fever. On physical examination, a slight systolic bruit was heard at the apex. The area of cardiac dulness appeared to be slightly increased, and the heart s impulse somewhat diffused. A remarkable crop of subcutaneous nodules, varying from the size of a pea to that of a large cob-nut, was discovered, and proved a striking feature of the case. They were largest and most abundant on the scalp, but were conspicuous also on the back, on the extensor aspect of the hands and fingers, and on the knees, ankles, and feet. During the first few days after admission some of the nodules began to subside, and fresh ones appeared; a presystolic murmur developed, with thrill; then a slight double pericardial friction-sound. The temperature ranged between 98° and 100° F. A third crop of nodules appeared in January, six weeks after admission, but after this they ceased to come out, and gradually disappeared. The cardiac murmurs declined, and at the beginning of March the boy was discharged convalescent, the double mitral murmur still being audible. He was, however, readmitted on April 26, for some slight joint-trouble and increased malaise. Fresh crops of nodules appeared, the temperature went up to from 99° to 101° F., choreic movements developed, and remarkable emotional excitability. He would cry at a word, or without reason. The heart's action became excited and irregular, running up to 130 and 140. This rapid, excitable action continued from this time to be one of the most prominent features. Pericardial friction was again heard, and another crop of nodules appeared shortly after. The pallor and weakness increased; the heart's action continued rapid, in spite of the free administration of digitalis. Slight dropsy appeared, the heart's action became more rapid and feeble and soon failed altogether, death taking place just nine months after his first admission. During the whole of this long period the endocarditis and pericarditis revived from time to time, fresh crops of nodules continued to appear, anæmia increased, and, in spite of complete rest, salicylate of sodium, salicin, alkalies, quinine, iron, digitalis, and opium, the disease was practically unchecked and ran its course to a fatal issue with but slight remission. The articular affection was throughout only slight and occasional, being usually entirely absent.

Post-mortem examination showed some pleuritic adhesions and an enormously thickened pericardium adherent throughout. The right auricle was dilated, the walls of the right ventricle thin and pale; the tricuspid valve was covered with fine granulations almost entirely on the auricular side. The pulmonary artery was considerably dilated. The left auricle was dilated. The left ventricle was considerably dilated, the mitral valve remarkably thickened, especially at the edges on the auricular aspect, and there were numerous non-vascular granulations; the chordæ tendineæ were shortened and much thickened, the musculi papillares hypertrophied and tough. The cusps of the aortic valves were all more or less thickened around the edges, the anterior one having a well-marked dilatation towards the ventricle about the size of a split pea. The lungs showed marked collapse in certain portions, but no pneumonic change.

This case is a representative one of persistent progressive rheumatic endocarditis and pericarditis, with accompanying evolution of nodules in successive crops, progressive anæmia and wasting, rapid cardiac action, and finally death from cardiac failure.

Another characteristic feature of endocarditis in children, at any rate of the rheumatic form, is anæmia. It is most marked in the protracted and relapsing cases, and may be due partly to the effect of the rheumatic poison, but partly also, I think, is attributable to the imperfect circulation through the pulmonary vascular system. For this anæmia is as much a feature of mitral disease in children as it is of aortic regurgitation in adults. In children the turgid, congested face of mitral stenosis and regurgitation is rarely seen,—but, in its place, pallor. With the anæmia often progressive there is also in some cases wasting: the child grows thin and feeble.

Again, in the relapsing endocarditis of children producing serious mitral disease, hypertrophy is set up, which proceeds rapidly and sometimes attains enormous dimensions, with some dilatation, but dropsy seldom follows. It is rare to see a child waterlogged from heart-disease. Renal dropsy is common, cardiac dropsy rare. When endocarditis is directly fatal, it is usually in association with pericarditis, or hypostatic pneumonia, or embolism, and death results from anæmia and heart-failure. The reason is, no doubt, that in the growing tissues of children compensatory hypertrophy is easily set up and well maintained, and dilatation is seldom extreme.

In a certain proportion of cases, whether rheumatic or not, where the inflammation is more acute or where it attacks afresh structures previously damaged, distinct cardiac disturbance and general symptoms mark its onset. The child is restless, uneasy, and looks distressed; there is a sense of discomfort in the præcordial region, palpitation, a quickened, excitable pulse, a rise of temperature, perceptible early, marking some fresh cause of disturbance, even in cases where the febrile state of rheumatism or other underlying disease already exists. It is a question, however, whether these more pronounced symptoms are not due to extension of inflammation to the pericardium or the muscular tissue. For cardiac symptoms are most prominent in those instances where endocarditis is complicated by pericarditis and by myocarditis. The occurrence of the former would be indicated chiefly by the development of friction-sound, by signs of effusion, by dyspnœa and distress, and by quickened, enfeebled pulse; the advent of myocarditis, by irregular action of the heart, a feeble, uncertain pulse, dyspnœa, and sometimes dropsy from rapid dilatation of the softened, enfeebled walls.

Embolism is an occasional result of endocarditis; sometimes thrombi form during life in the feeble right auricle, causing great embarrassment of its action, which becomes excited and irregular. Detached fragments may be carried into one of the branches of the pulmonary artery, but this occurs most frequently when there is pericarditis also. This accident is usually indicated by a rise of temperature of two or three degrees, increase in the pulse- and respiration-rate, and physical signs of pneumonia in one or more limited patches of small area. Or there may be sudden embolism of the left middle cerebral artery, causing hemiplegia, from detachment of a particle of fibrin from a mitral vegetation, or signs of infarction of the spleen. I have had a case of this kind under my care, where the sudden access of pain in the region of the spleen, with the development of a tender splenic tumor there, and a wave of pyrexial disturbance, were the first indications of the serious nature of a mitral murmur previously judged to be hæmic and unimportant.

In certain cases, again, ulceration occurs, and the symptoms of septicæmia are added to those of endocarditis. This form of the disease is, however, of sufficient interest to claim a brief separate notice.

As results of the damage to the cardiac valves, serious obstruction by large vegetations, or from ulceration or perforation of a valve-segment, may occur; but I have not observed this in the case of children.

In mitral disease, when the heart begins to flag, and the valve-lesion is considerable, the pulmonary congestion to which this gives rise is liable to set up pleurisy and more or less extensive subacute lobar pneumonia of the bases of the lungs.

Diagnosis.—The diagnosis of endocarditis practically turns upon the existence of a murmur or other changes in the normal heart-sounds; and the paramount importance of making a careful examination of the heart in children in all cases in which endocarditis might arise, previously urged, must again be insisted upon. This should never be omitted in any affection connected with rheumatism, however trivial, such as slight joint-stiffness or tenderness, chorea, tonsillitis, erythema, or an unexplained febrile attack. Of these morbid cardiac sounds by far the most common is a murmur with the systole, audible in maximum intensity at the apex. Taking this systolic mitral or regurgitant murmur first, it may probably be produced in several distinct ways: by endocarditis causing thickening of the valve-flaps, and, through this, imperfect closure and leakage; by similar incompetence caused by muscular debility the result of myocarditis, or of pyrexia, or of anæmia. The decision as to which of these is the real cause of the systolic mitral bruit will depend upon several considerations. In the first place, the period of the attack at which the murmur is developed affords important evidence. Dr. Sansom[1] points out that the systolic apex-murmur in rheumatic fever, at all events, is generally developed early in the attack,—not late, as in the functional murmurs of typhus and typhoid: it is therefore probably not produced by the same cause. It cannot be anæmic, because in a primary attack it is developed before the anæmia, and if it were caused by anæmia there ought to be developed at the same time a pulmonary hæmic murmur; but this is not found, as a rule; if it does appear, it appears later. It has been assumed that if this murmur disappears it is functional, not organic. But the murmur is the same in character, and in time and mode of gradual onset, whether it remains or disappears. Sometimes the murmur dies down and reappears again and then remains permanent. It is much more reasonable to suppose that the mitral murmur which disappears is organic, like the murmurs which remain, than to suppose that a special hæmic murmur, unknown in the early stages of other acute diseases, should in these particular instances be developed at the mitral orifice, where, to say the least of it, functional murmurs are rare. The difference is probably that in the one case the valvulitis subsides without doing permanent damage, in the other it remains.

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[1] Lettsomian Lectures, p. 18.

Reasons were given, in discussing the etiology of endocarditis, for believing that the systolic murmur developed in chorea is usually organic, and probably always in that case the result of rheumatic endocarditis.

The early systolic murmur, then, is almost certainly organic,—due either to inflammatory affection of the valves causing thickening and leakage, or possibly in some cases to myocarditis causing muscular relaxation and leakage. Looking to the resulting valve-changes found post mortem, it must be judged to be most commonly the former; but, be this as it may, the immediate cause of the murmur is endocardial inflammation. A mitral systolic murmur, then, of recent inception, occurring early in the course of rheumatism or pyæmia or scarlet fever, and generally in chorea, must be considered almost certain evidence of the advent of endocarditis.

A presystolic murmur is always organic, and therefore its fresh appearance would be conclusive of the existence of endocarditis, past or present. It is, however, not quickly developed: it is some time, apparently, before the rigidity and narrowing are sufficient to produce the characteristic rumble and thrill. In the early stage it exists as reduplication of the second sound audible at the apex only, and sometimes accompanied by a bruit following the reduplication, as previously described. This special form of reduplication of the second sound may, I think, be regarded as distinctive of mitral valvulitis, which usually results in stenosis. The reduplication may disappear; but in the vast majority of cases it persists and is gradually changed into the presystolic rumble.

An *aortic systolic murmur* is almost invariably organic. Exception must be made in certain cases of extreme anæmia, where it appears as a functional hæmic murmur, in place of the ordinary pulmonary bruit or in conjunction with it. Yet, as with the mitral murmur, so with the aortic systolic murmur: if it occurs early, or without sign of marked anæmia, it must be regarded as organic.

A *diastolic aortic murmur* is invariably organic: there is no exception.

It occurs sometimes, although rarely, as the earliest sign of endocarditis. In one instance I watched its gradual development in a child of strong rheumatic predisposition, in whom it reached its full height before any sign of rheumatic arthritis appeared. This came a fortnight later, and consisted in a slight tenderness and swelling of one wrist. A year afterwards the child had general articular rheumatism.

A *pulmonary murmur* is never organic unless of congenital origin, and affords, therefore, no evidence of endocarditis.

A *tricuspid regurgitant murmur* arising in a case where there was no evidence of previous disease leading to dilatation would be almost conclusive proof of endocarditis. Exception must be made, however, with regard to those cases of simple dilatation which arise after certain acute diseases, particularly scarlatinal nephritis, apparently without endocarditis, from simple giving way of the enfeebled cardiac muscle under the stress of increased resistance from uræmic vascular spasm.

Accentuation of the second sound has some value as evidence of endocarditis. It shows increased resistance in the pulmonary vessels, of which mitral regurgitation or obstruction causing pulmonary engorgement is a common cause; but it does not afford absolute proof that the mitral defect is organic. Yet, if the accentuation is very marked, it is always due, I think, to organic disease. A heart with paretic enfeebled muscle does not contract vigorously enough to produce such marked recoil. When signs of endocarditis such as those mentioned arise, it is sometimes difficult to determine whether they are set up by recent inflammation or are caused by permanent valvular changes the result of some former endocarditis. If the state of the heart is known to have been normal previous to the attack, the endocarditis must be recent; but if the condition of the heart is either not known or is known to have presented signs of previous valvular disease, the existence of present endocarditis can be determined only by other evidence.

The character of the murmur is some guide: if soft and blowing, it is probably recent; if harsh, vibrating, or musical, it is probably of older standing. Yet this test is by no means to be depended on, for I have twice lately observed a murmur which when first discovered was so soft and gentle as to render its actuality a matter of doubt and discussion, become in the course of a single week harsh, coarse, and musical.

The existence of hypertrophy and dilatation, the presence of dyspnœa or of pulmonary congestion or dropsy, or a history of previous rheumatism, would tend to support the view of an old lesion. Yet it is to be remembered that the existence of an old endocarditis, instead of being prohibitive of fresh attack, renders it more likely. If acute articular rheumatism is present, the advent of fresh endocarditis must be regarded as highly probable. The occurrence of any aggravation of cardiac symptoms or intensification of the murmur is likewise suggestive of its recurrence.

Prognosis.—The view to be taken of the future course of endocarditis and its results must depend in some degree upon the character of the acute rheumatism, scarlatina, or septicæmia which has given rise to it.

The condition of the heart before the attack forms also a serious element in prognosis. If the heart was intact up to the time of the development of the murmur, the immediate prospect is usually favorable. In children especially it is rare for a first attack to be fatal. But if old-standing heart-disease already exists, if there be great hypertrophy and dilatation, the prospect is far more grave,—and grave in proportion to the previous mischief. In the case of children the prolonged relapsing form of endocarditis associated with rheumatism, lasting in almost continuous form for months and little influenced by treatment, is always serious, and the appearance of nodules from time to time in successive crops renders the prognosis still more unfavorable. Other unfavorable signs are progressive anæmia, wasting, and rapid, feeble action of the heart. The complication of pericarditis or myocarditis, of pleurisy or pneumonia, of cyanosis, of dyspnœa, adds greatly to the gravity of the outlook.

If, on the other hand, the cardiac murmur subsides during convalescence and does not reappear, if there is no succession of relapses, if the anæmia disappears and the child keeps up flesh and vigor, there is good hope of perfect recovery. Even when the mitral defect remains, compensation is so readily effected in children that, if the damage is not extreme, comparatively little evil result may follow.

On account of the rapid growth of tissue in early life, the hypertrophy proceeds at a great pace, and the result appears to be good or evil according as the original lesion of the valve is slight or extensive.

As with adults, the most serious valve-lesion is that of aortic regurgitation; next to this comes mitral stenosis, followed, in order of gravity, by mitral regurgitation and aortic obstruction.

Treatment.—Something may be done to ward off an attack of endocarditis in those diseases in which it is liable to arise as a complication,—such as rheumatism, chorea, scarlet fever, measles, puerperal fever, pyæmia, and septicæmia. The relief of this underlying condition as promptly as possible may reasonably be expected to lessen the chance of the heart becoming implicated, although this has less influence than might be expected, owing to the fact that the heart-affection usually occurs early. It has been shown in the article on rheumatism (vol. i. p. 816) that salicin and the salicylates have no proved power in lessening the liability to heart-disease in that affection; yet if acute arthritis and pyrexia are present it will be well to arrest them quickly at the outset by these remedies, if possible before endocarditis appears. For, even although they may be too late to prevent an early endocarditis, and do not apparently modify it favorably when it is actually established, they probably, by shortening the duration of the rheumatic state, lessen the liability to endocarditis later.

Two other precautionary measures should also be taken in all conditions possibly productive of endocarditis,—protection against chill, and the maintenance of absolute rest, both mental and bodily. Chill favors internal congestions and the production of the rheumatic virus; exertion of all kinds increases the force and frequency of the heart's action, and thus causes increased flow of blood to and increased shock and friction of the valves, favoring therefore the development of inflammation.

When endocarditis has actually arisen, the means at our disposal for relieving the inflammation are, unfortunately, extremely limited. The lining of the heart lies practically out of reach; we cannot act upon it, as we can upon the parietal pericardium or pleura, through the connection of its vessels with those of the neighboring surface. The only means by which the circulation in the inflamed parts could be reached would be by general bloodletting, or by remedies, such as tartar emetic or aconite, which act as cardiac depressants. Anything which seriously enfeebles the action of the heart is theoretically objectionable, especially with children; and indeed the actual results of such treatment have proved highly unsatisfactory, and even disastrous.

The chief point, again, is to give the heart as much rest as possible. It is as necessary as a means of relief when endocarditis is established as it is as a prophylactic against it. Dr. Sibson's observations[1] showed that, although absolute rest only slightly diminished the proportion of cases of endocarditis in acute rheumatism, yet it modified remarkably the extent, severity, and permanent ill effects of the valvular inflammation. It is reasonable to suppose that rapid forcible action would increase the irritation of the valve-structures and augment the flow of blood thither, and thus aggravate existing inflammation; and the direct evil results which follow cardiac strain or excitement sufficiently confirm it. The following case illustrates this point, as well as some other points of importance:

H. G., a boy of fourteen, was brought for advice on account of hard nodules on the palms of the hands, attributed to gout: they caused so much stiffness that he could hardly use his fingers. Some along the flexor tendons were oval and the size of almonds, and there were smaller ones on the wrists, elbows, and chest. Although in an unusual situation, they were judged to be rheumatic. The boy's sister had had chorea followed by scarlatina, his grandmother rheumatic fever, but he himself had never had anything like acute articular rheumatism or chorea or scarlatina. On cross-examination, however, it appeared that he had had some tender swelling and stiffness of the wrists and knees, which the doctor had diagnosed as rheumatic; but the state of the heart was not examined. Since then the boy had failed remarkably in his capacity for physical exertion. He was at a large public school and a leader in all athletic sports and was especially good at foot-races. Latterly he found he could not run: he was always beaten, and greatly exhausted. For similar reasons he had given up foot-ball.

On examining the chest, a loud aortic regurgitant murmur was heard; pulsation was visible over the fourth, fifth, and sixth spaces, the apex-beat outside the nipple in the sixth space; dulness extended to mid-sternum. There was clearly extreme dilatation of the left ventricle. The pulse was collapsing; the carotids pulsated obtrusively in like manner.

Here, then, was the secret of the boy's failing aptitude for athletic exercises. The mischief, had, no doubt, commenced in the rheumatic attack sixteen months before. The disastrous result was due to continuance of violent physical exertion,—constant excessive cardiac strain. Had the heart been examined at the time, the endocarditis would have been discovered, perfect rest enforced, and complete recovery might possibly have taken place, instead of permanent and ultimately fatal injury.

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[1] Reynolds's System of Medicine, Eng. ed., vol. iv. p. 527.

In endocarditis, then, physical and mental rest must be alike enforced, for mental excitement quickens cardiac action, and the patient must be kept in bed or at rest on a couch long after all signs of active disease have subsided. For the same reason, the diet must be easily digestible, simple, and unstimulating. A full meal of solid food excites the circulation, and nourishment should therefore be limited to milk, beef tea, and light farinaceous preparations. Alcoholic stimulants should be avoided unless demanded by cardiac failure.

In rheumatic cases salicylate of sodium should not be given if endocarditis has already arisen. As was shown before, it appears to have no power in controlling endocarditis, and its depressing action on the heart renders it injurious. It should be at once stopped, therefore, if it is being administered at the time. If, however, articular rheumatism is present, salicin, which has little or no depressant property, may be given in doses of five to seven grains every four hours for a child five years old, in water sweetened with syrup of orange. To the salicin may with advantage be added an alkali, such as the citrate of sodium, the sodium salts being less depressant than those of potassium. The treatment of rheumatism by alkalies, as stated by Dr. Fuller and Dr. Dickinson, appears to give more favorable results than any other, so far as cardiac inflammation is concerned. The citrate or carbonate of sodium may be given in doses of ten grains every four hours until the urine becomes slightly alkaline, and the amount regulated afterwards so as to keep it in this condition.

If the temperature runs high, quinine should be given in full doses: one to three grains may be given every four hours to a child of five. The acid hydrobromate is the least irritating: it causes less sickness than the sulphate, and has the advantage of extreme solubility without acid, so that the dose may be given in small compass,—in a teaspoonful of water well sweetened with syrup. In case of difficulty in giving it by the mouth, it may be administered in the form of enema in five-grain doses. When the endocarditis is rheumatic, alkalies should be administered with the quinine. They may be given separately or together,—six to ten grains of the citrate of sodium with two grains of quinine and ten of citric acid and half a drachm of syrup of orange-peel to half an ounce of water.

In all septic cases quinine should be given freely from the first, with abundant nourishment of the most concentrated kind, such as strong meat teas and essences, and milk. Peptonized food is likewise useful, for it is a question whether in these cases the digestive apparatus duly performs its function. In septic endocarditis an exception must be made with regard to stimulants. Alcohol is so good an antiseptic that its influence in this respect probably more than counterbalances any evil which may accrue from its effect on the circulation in the valves.

When the action of the heart is rapid, tumultuous, and excited, yet feeble withal, as it so often is in endocarditis supervening on old valvular disease or when accompanied by pericarditis, digitalis, in doses of three to five drops of the tincture every four hours, or half a drachm of the infusion, has usually a remarkable sedative and tonic effect, slowing the beats and increasing their fulness and regularity. In some cases, especially those where pericarditis has caused much thickening and close adhesion of the pericardium, or where there is great hypertrophy, digitalis aggravates the palpitation and causes increased faintness and distress. Then opium is often effectual in soothing the heart's excitement, slowing the pulse, and relieving distress. It may be given in doses of one to three minims every four hours to a child of five years, its effect being closely watched, so that the dose may be at once reduced if it should produce too great drowsiness or pulmonary embarrassment.

Strophanthus, in doses of one to two minims of the tincture every four hours, is also a useful heart sedative and tonic. In my experience, however, it has proved inferior to digitalis and to opium in the rapid action and heart-failure of endocarditis.

ULCERATIVE ENDOCARDITIS.

Endocarditis sometimes assumes a malignant form, with symptoms in some cases of a typhoid, in others of a pyæmic or septicæmic character: in the latter the pyrexia has the hectic type, accompanied by rigors, profuse sweatings, more or less diarrhœa, sometimes jaundice, rapid pulse, and great prostration. The spleen enlarges, and embolic infarcts may occur there and in other organs. In some cases there is a hemorrhagic or erythematous or papular rash, or all three together, which has been mistaken for that of typhus or small-pox; sometimes there is delirium or coma, sometimes acute meningitis; usually, but not always, a distinct murmur, mitral or aortic, can be detected. This form of endocarditis has invariably proved fatal in all cases yet recognized. After death soft fungoid vegetations are found on the cardiac valves, sometimes suppurating; and generally, but not invariably, there is ulceration. Abundant micrococci are found in the vegetations; but whether these have any specific character, and what is the exact part which they play in the development of the disease, are questions which remain as yet unsettled.[1]

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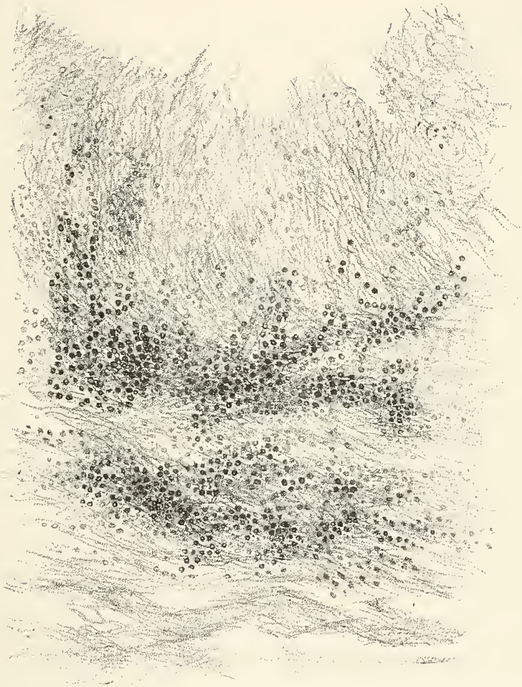
[1] The observations of Wechselbaum, Wyssokovitsch, Hirschler, and Stern show that while the staphylococcus pyogenes aureus et albus and streptococcus pyogenes constitute the etiological element in most cases, yet other and various forms of micro-organisms may induce the same ulcerative changes. Ann. Univ. Med. Sci., 1888, vol. i. p. 187



SECTION OF AORTIC VALVE IN ULCERATIVE ENDOCARDITIS.—Showing similar proliferation and cell-infiltration of sub-endothelial fibrous tissue.

This malignant form of endocarditis arises in two distinct ways,—as a disease of the valves or of the endocardium, either primarily or in connection with acute or subacute rheumatism, scarlet fever, diphtheria, or other specific fever, especially where there is old-standing valvular disease, or acute disease, such as pneumonia, when the valve-lesion is the disseminating centre of infection; and also as part of a general pyæmic or, as Rosenstein holds,[1] diphtheritic condition, either by inoculation through an open wound or from a puerperal source.

Ulcerative or malignant endocarditis is a comparatively rare disease, although many cases are no doubt erroneously classed as typhoid or other form of malignant fever.

Prof. Osler, who has recently so ably reviewed[2] this subject in his Lectures at the Royal College of Physicians of London, found records of upward of two hundred cases. The majority of them appear to be in young people under thirty; yet it is seldom seen in children, probably because it is so especially connected with degenerating influences, such as alcoholic excess, want, and exposure. The earliest case, that recorded by Dr. Kirkes, who first recognized the disease, was in a boy of fourteen. Prof. Osler (*loc. cit.*) gives a case in a boy of eleven associated with chorea. Dr. Ord[3] cites one in a girl of sixteen who had had neither rheumatism nor chorea nor scarlet fever.

Only a single case appears in the records of the Hospital for Sick Children during the last twenty years, where patients are admitted under the age of twelve. The child was a girl of eight, and the youngest case that I can find yet recorded. She had suffered from acute articular rheumatism three years before, and some two years later was in hospital for chorea. From this she soon recovered, and remained well until five weeks before admission, when she had incessant vomiting and headache, followed by an attack of general convulsions. Twitchings and unconsciousness lasted twelve hours, but no paralysis remained. Three days afterwards another attack of convulsions occurred.

When admitted into hospital she was suffering from great dyspnœa and had to be propped up in bed. The respirations were 60, the pulse 132, the temperature in the axilla 104.2° F. The complexion was externally pallid, with a greenish tinge, but there was no actual jaundice. There was no œdema nor dropsy of any kind. The cardiac region was bulging, with heaving impulse reaching outside the nipple to the sixth space, and a large area of cardiac dulness. There was a prolonged systolic apex-murmur. A few râles could be heard at the base of the lungs. The liver and spleen were not enlarged. The urine contained a trace of albumen.

The same evening a fresh attack of convulsions came on, especially of the right arm, with squinting, contracted pupils, and almost complete unconsciousness. The following day consciousness and speech returned, but the left arm and leg and left side of the face were found to be completely paralyzed. The pulse rose to 158. Respirations were 56; temperature, 103° F. She remained in much the same state for four days; then complete insensibility came on, and death took place on the sixth day after admission.

On post-mortem examination, the pericardium was found firmly adherent throughout; the heart greatly hypertrophied, weighing twelve and a quarter ounces. The left auricle

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[1] Ziemssen's Cyclopædia, vol. iv. pp. 65, 66, 70.

[2] Lancet, March 7, 1885. p. 415.

[3] Ibid., 1888, vol. i. p. 724.

was much dilated and its lining membrane opaque, and just above the aortic segment of the mitral valve was composed of thickened endocardium with adherent lymph attached in polypoid masses, and sharply-cut ulcers owing to breaking down of atheromatous-looking patches just above the root of the flaps at their junction. The mitral valve was greatly thickened and shortened, and polypoid vegetations were attached, but there was no ulceration on the flaps themselves. Infarcts were found in the kidneys, spleen, and right middle cerebral artery.

In ulcerative endocarditis treatment is seemingly useless. But the condition is not always to be diagnosed with certainty, and it is right to give remedies which tend to counteract the septic condition. Quinine in full doses, concentrated liquid nourishment in small quantities at short intervals, with a free administration of stimulants, and, if necessary, opium, are the chief measures which afford a possibility of relief.

ENLARGEMENT OF THE HEART.

By J. MITCHELL BRUCE, M.D., F.R.C.P.

Definitions.—The term "enlargement" is applied (1) to certain processes by which the heart increases in size, and (2) to various conditions of the organ which are the results of these processes.

(1) Regarded in the first of these two senses, cardiac enlargement is of three kinds, which may thus be defined:

Hypertrophy.—A process of general uniform enlargement of one or more of the chambers of the heart, which consists in increase of its muscular tissue, and leads to thickening of the walls. It is always reparative or compensatory in its effect.

Dilatation.—A process of general uniform enlargement of one or more of the chambers of the heart, which consists in overstretching of the elastic structures of the walls, and leads to increase of its capacity. It is of two entirely different kinds. The first kind is *dilatation from overfilling* of a chamber, and is compensatory in its effect; the second kind is *dilatation from incomplete emptying* of a chamber, and is always associated with failure or inadequacy of the cardiac force.

(2) The various conditions of the heart which result from these three processes are found to be the following:

Compensatory dilatation with hypertrophy;

Simple hypertrophy;

Either of the above two conditions in association with dilatation from failure;

Simple dilatation from failure.

The combinations of hypertrophy and dilatation are also described as "dilated hypertrophy" and "eccentric hypertrophy."

Hypertrophy, dilatation, and dilatation with hypertrophy may be either (1) general,—that is, involving the whole heart,—or (2) partial or local, when the change does not extend to all the chambers.

Pathological Anatomy.—When we proceed to inquire into the existence of enlargement of the heart in children, it is necessary to bear in mind the absolute measurements and weight of the normal heart, as well as the relative weight of the heart and body at different ages. These facts are set forth in the following tables:

TABLE I.

Showing the Measurements of Length, Breadth, and Thickness, in Centimetres, of the Left Ventricle in Children at Different Ages.[1]

Boys.						Girls.					
Age, Years.	Length.	Breadth.	Thickness.			Age, Years.	Length.	Breadth.	Thickness.		
			Base.	Middle.	Apex.				Base.	Middle.	Apex.
1–4	5.14	6.09	.67	.65	.43	1–4	5.10	5.83	.57	.63	.46
5–9	7.04	7.44	.74	.86	.58	5–9	6.0	6.54	.69	.70	.52
10–15	7.67	8.35	.81	.86	.52	10–15	6.59	7.04	.74	.72	.54

TABLE II.

Showing the Average Absolute Weight, in Grammes, of the Heart in Children at Different Ages; with the Relative Weight of the Heart to the Body-Weight.[2]

Age.	Absolute Weight.		Relative Weight.	
	Male.	Female.	Male.	Female.
At birth	20.79	19.24	.00620	.00629
One month	16.19	14.36	.00643	.00632
Second to sixth month	20.13	20.18	.00576	.00610
Seventh to twelfth month	30.64	32.14	.00597	.00602
Second to third year	52.7	45.2	.00615	.00616
Fourth to fifth year	65.2	69.0	.00580	.00591
Sixth to tenth year	103.6	82.5	.00623	.00561
Eleventh to fifteenth year	163.8	177.4	.00600	.00551

It must not be forgotten that the relative thickness of the walls of the two ventricles is in the new-born child different from what afterwards obtains. At birth the left ventricle measures from .44 to .68 centimetre, the right from .34 to .44 centimetre. Up to the sixth year the left measures not quite one centimetre, the right from .3 to .4 centimetre, the thickness of the right ventricle rapidly declining after birth, until in the sixth year it is scarcely so great as in the new-born.[3]

In pure or simple *hypertrophy* of the ventricles the thickness of the wall is increased, until it may exceed the normal by one-quarter or one-half, or may even attain double the natural measurement. The columnæ carneæ are proportionately or even disproportionately robust. The weight is always increased. Hypertrophy of the right ventricle, though great, may be less striking than hypertrophy of the left, and is rarely found pure post mortem. Hypertrophy of the walls of the auricles demands close examination for its detection: it is probably never pure. Either auricle may reach double the normal thickness. The muscular tissue in hypertrophy is of a brownish-red color, and peculiarly firm to the finger; the walls preserve their concentric outline when incised.

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[1] Bizot, quoted by V. Dusch, in Gerhardt's Handbuch der Kinderkrankheiten, 1878, iv. 1, 269.

[2] W. Muller, Die Masse des menschlichen Herzens, 1883, p. 56. It is necessary to remember that different observers have adopted different methods of preparing the heart in estimating its absolute and relative weight. Some removed the whole of the great vessels; others left one inch of each in connection with the heart. Some removed the pericardial fat, to obtain a correct estimate of the myocardium; others did not. The maximum and minimum weights range widely on either side of the averages given above.

[3] Von Dusch, op. cit., p. 269.

In pure or simple *dilatation* of the heart the enlargement is found to be due to increased capacity of one or more of the cavities, with thinning of the walls. Dilatation is usually determined by observing the unnaturally globular shape of the organ as a whole, the visible enlargement of one or more of its cavities when opened, the great bulk of the clot within it, the flattening of its columnæ carneæ, and the more sac-like, rounded appearance which it presents when emptied and freely exposed to view. A more exact estimate is made of the degree of dilatation by measuring the maximum length and breadth of the opened chamber and the thickness of the parietes, the latter being comparatively diminished. The muscular tissue is variously altered in color, and so reduced in firmness that the walls collapse on section. In pure dilatation the weight of the heart is never increased.

Dilatation with hypertrophy presents a combination of the characters of the two simpler forms just described. The degree of enlargement is, as a rule, greater than in either of these. It proves to be due partly to increased capacity of one or more of the chambers, partly to an increase of the muscular tissue, which is sufficient to augment, preserve, or nearly preserve, in different instances, the normal thickness of the walls in the presence of the dilatation. Great variety occurs in the relative degrees of the two associated changes, with corresponding differences in shape and weight of the heart. The weight of the organ is always increased, and may reach that of the normal adult heart. When the dilatation is consequent on failure, the walls are peculiarly pale, soft, or yielding to the finger, and may be so flaccid as to collapse entirely on section.

In the great majority of instances the heart is also the seat of disease of the valves or pericardium, which is regarded as the primary lesion.

Histology.—Hypertrophy of the heart consists in a true increase in size of the individual muscular fibres, accompanied by an increase in the number of these elements (hyperplasia). The histological appearances of the myocardium, apart from these changes in size and number, are perfectly normal.

In *dilatation* the tissues of the walls may not present any abnormal characters under the microscope. More frequently they are found to be pigmented, or in a condition of granular, fatty, or fibroid degeneration, or of acute or chronic myocarditis.

Condition of the other Viscera.—The condition of the other great viscera —the lungs, liver, spleen, kidneys, and alimentary canal—and of the central nervous system varies considerably with the primary lesion. Independently of this, when dilatation from failure has set in, passive hyperæmia or mechanical congestion is always found in these organs; and if this continue long, or be frequently repeated, slow pigmentation and fibroid degeneration, with wasting of the parenchyma, make their appearance. With these visceral changes there occur effusion into the cavities of the peritoneum, pleuræ, and pericardium, and dropsy of the cellular tissues, catarrhs of mucous surfaces, and occasionally hemorrhages from venous rupture or erosions.

Etiology.—In most cases of cardiac enlargement in children the cause of the change consists in primary lesions of the heart or larger arteries. By far the most common of these lesions is valvular disease. Next in order of frequency as a determining factor of enlargement is adherent pericardium. Congenital disease of the heart and great vessels is a cause of cardiac enlargement almost peculiar to the period of infancy and childhood. The earlier the age, the more probably is malformation the primary lesion, until in the infant all other causes may be practically disregarded.

Much less striking than these gross changes or imperfections, but of equal importance in determining enlargement of the heart, are certain morbid conditions of the myocardium. Such are the granular degeneration of the muscular fibres that occurs in typhoid fever, scarlatina, diphtheria, and other acute specific diseases, and the myocarditis of acute rheumatism and pyæmia, all of which may be the cause either of acute primary dilatation or of a secondary dilatation supervening on previous enlargement. Failure of nutrition from coronary disease is practically never seen in children. Fatty degeneration is very rare.

In other instances of this "secondary" dilatation the microscope may fail to detect any histological change in the myocardium, but a careful study of the child's history reveals as the cause of dilatation the existence of serious interference with the conditions necessary for healthy cardiac nutrition. The circumstances that induce this unfavorable effect are extremely various. Some of them tell upon the myocardium through the medium of the blood, or it may be the nervous influences on which the nutrition and healthy activity of the heart constantly depend. Such are anæmia, dyspepsia, poverty, unhealthy social surroundings, the abuse of rest, the demands of rapid growth and development; or it may be a combination of these. Acute and subacute rheumatism—especially that persistent or recurrent, possibly latent, type so common in the child—is one of the most frequent, intractable, and serious causes of malnutrition and secondary dilatation of the walls of the enlarged heart in chronic cardiac disease. Acute pulmonary diseases have a similar effect in debilitating the heart and permitting dilatation. Nervous disturbances, such as the intellectual strain and anxiety connected with schooling, undue excitement even of a pleasing character, and chorea, also promote the occurrence of this kind of dilatation in children who are already the subjects of chronic valvular disease.

In a smaller number of cases of hypertrophy and dilatation the cause has to be searched for in some disturbance of the circulation entirely apart from the heart. Enlargement of the right ventricle in children is frequently referable to chronic pulmonary disease, which increases the resistance to the passage of blood through the lungs. The abnormally high arterial tension of Bright's disease may give rise to enlargement of the left ventricle, particularly pure hypertrophy, but this is uncommon in the child (Dickinson). Acute dilatation of this chamber occasionally results from renal congestion in scarlet fever;[1] and this is liable to occur if the heart have been previously enlarged. Arterial degeneration, an increasing source of cardiac enlargement as age advances, is exceedingly rare in children. The same remark applies to protracted functional excitement of the heart, —for example, in Graves's disease. Muscular exertion is met with as a cause of hypertrophy of the heart in boys who have been allowed to indulge too freely in running and other games and athletics. Rapid dilatation of the right ventricle may take place in whooping-cough and other diseases, such as croup, proving fatal by asphyxia.

Pathology.—We have now to inquire into the nature of the process by which the conditions and circumstances that have been traced into etiological connection with enlargement of the heart give rise to these remarkable alterations in the thickness of its walls and the capacity of its chambers.

Hypertrophy.—The origin of hypertrophy of the heart is to be found in two physiological laws of capital importance. The first of these laws is that the force displayed by a muscle or muscular organ in contraction is in proportion to the weight or load that it has to lift; that the heavier the load (always within a certain "reasonable" limit), the more forcible or vigorous will be the muscular contraction; in other words, that a muscle under ordinary circumstances possesses a certain reserve of force against extraordinary demands. The second of these laws is that when a muscle or muscular organ displays more than the ordinary amount of force for a considerable period of time it increases in size, provided it enjoys sufficient nutrition.

In the case of hypertrophy of the heart, the muscular organ is the wall of one or more of the chambers. The weight or load is measured by the intracardiac pressure or tension during systole, and consists of the charge of blood within the chamber, which has to be moved forward by the muscular effort into the next portion of the circulatory apparatus—whether from the auricle into the ventricle or from the ventricle into an artery—against an increasing resistance. If the resistance to the forward movement of the blood is unnaturally raised, the muscular wall of the chamber of the heart concerned in the movement will first act more vigorously and then in course of time become hypertrophied. The nature of the process may be more readily comprehended by examining an instance.

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[1] Goodhart, Guy's Hosp. Rep., 1879, vol. xxiv. p. 153; Barlow, Med. Times and Gaz., 1880, vol. i. p. 426; Silbermann, Jahrb. f. Kinderkr., 1881, xvii. 182.

The development of *hypertrophy of the left ventricle*, in a pure form, may be studied in obstructive diseases of the aortic valves. This lesion, which in children is commonly the result of endocarditis, presents a certain obstacle to the passage of blood in systole from the left ventricle into the aorta. To speak more correctly, it increases the chief part of the work which the left ventricle has to accomplish in systole,—that part, namely, which consists in forcing open the aortic valves and discharging the ventricular contents into the aorta. The opening of the aortic valves and the penetration of the aorta (as it is conveniently called) by the discharged volume of blood are accomplished by the left ventricle against the arterial or blood pressure within the aorta, which weighs the valves down and opposes the influx of blood from the heart. Now, it is obvious that if the aortic valves be stiffened or completely fixed by inflammatory changes, or if the aortic mouth be narrowed, both the opening of the valves and the penetration of the aorta will be more difficult, the pressure within the left ventricle at the commencement of systole—that is, the resistance to the systolic contraction of the walls—will increase, and a greater display of force will be called for. Provided the difficulty be moderate, the muscular wall rises to the occasion by a display of its reserve force. If the valvular lesion be permanent, and the nutrition of the myocardium perfect, the left ventricular wall in course of time undergoes a pure increase in bulk of its muscular tissue,—an hypertrophy just sufficient to overcome the increased resistance ahead. The disability of the left ventricle is now perfectly removed; the circulation continues undisturbed; the physical evidence of a morbid condition at the mouth of the aorta—viz., a systolic aortic murmur—is accompanied by the physical signs of hypertrophy of the left ventricle which we shall presently describe; the subject of the valvular disease suffers from no symptoms of impaired cardiac function: we say that *compensation* of the circulatory defect is *established*.

Hypertrophy of the *right ventricle* in congenital obstruction of the mouth of the pulmonary artery arises in a manner precisely similar to that just described; and the same applies in cases of mitral disease and chronic lung-disease in the early stage while the reserve force is still sufficient.

It will be obvious to the reader from these considerations that hypertrophy of the heart is not a disease, but a natural method of recovery from disease, preventing or undoing its evil effects. If the aortic valves in a child are left diseased after an attack of endocarditis, we do not dread but welcome and encourage hypertrophy of the left ventricle, the means by which alone the balance of the circulation can be maintained. Further, whilst the aortic obstruction remains (most probably permanently), so long must the hypertrophy of the left ventricle continue. And if the hypertrophy threaten to fail, the welfare of the patient manifestly demands that it be restored by every means in our power.

Dilatation.—The first step in the development of dilatation of a cardiac chamber is over-distention with blood. A quantity of blood larger than ordinary is temporarily accommodated within the cavity *by virtue of the elasticity of its walls.* If this condition of over-distention be indefinitely repeated, or indefinitely prolonged, the walls become stretched, and the chamber that they enclose becomes correspondingly dilated.

It remains to be seen how excessive distention arises in connection with the causes of dilatation which we have traced.

The difficulties that attend this part of the subject appear to arise from confounding the two kinds of dilatation with each other. The first kind originates in excessive distention from overfilling consequent on valvular defect, and is invariably accompanied by hypertrophy, being, like it, compensatory. The second kind originates in excessive distention from incomplete emptying consequent on parietal weakness, and is either primary or supervenes upon the two other kinds of enlargement only when compensation fails. Whilst the physical result in the two kinds of dilatation is the same, their physiological significance is entirely different. A concrete example will make the subject more intelligible.

Dilatation from overfilling may be illustrated by the dilatation of the left ventricle (associated with hypertrophy) which is found in aortic incompetence. This lesion permits the regurgitation of a certain quantity of blood from the aorta into the left ventricle in each diastole. Besides this unnatural supply, there enters the left ventricle the regular measure of blood from the left auricle. The left ventricle, thus supplied from two sources, is overfilled by receiving an overcharge which it must, and does, accommodate,—an accommodation which can be effected only by stretching of its elastic walls. Continued over-distention becomes dilatation, a necessary and permanent condition of the ventricle as long as the valvular incompetence remains. This is dilatation from overfilling.

Concomitantly with the dilatation, hypertrophy is established. The over-distention of the ventricle is attended with increased internal pressure; the larger load that has to be driven stimulates the wall to more vigorous contraction and consequent muscular development, as already described; and compensatory dilatation with hypertrophy (the "secondary" hypertrophy of some authorities) is the complete result. Thus the process of dilatation from overfilling is, like hypertrophy, conservative or reparative in its effect. Given a serious lesion like aortic incompetence, dilatation of the left ventricle is a necessity if the circulation is to be maintained. The increased capacity of the chamber, referable to its reserve elasticity, provides a reservoir for the accumulation of blood behind the incompetent valve; and thus, along with the concomitant hypertrophy, the balance of the circulation is practically restored.

Dilatation from incomplete emptying of a cardiac chamber is always the result of inadequacy, absolute or relative, of the cardiac walls. Under the influence of one or other of the causes of dilatation enumerated under Etiology, an hypertrophied ventricle may not be able to maintain the increased display of force demanded of it by valvular or other lesion. We then say that the heart is "failing,"—that the "compensation has broken down." Symptoms of "heart-disease" make their appearance, and the physical signs of hypertrophy are now complicated with those of dilatation.

In order to understand how dilatation of the ventricle has been produced by failure of its muscular walls, we must bear in mind that, if the parietal energy be deficient, exhaustion occurs *before the completion of each systole* or unit of cardiac work. The force displayed by the heart in contraction wanes before penetration of the aorta has been perfectly effected, and a certain amount of the measure of blood that ought to have been discharged is left behind in the chamber.[1] To this residuum there is immediately added in diastole the ordinary charge of blood from the left auricle. The left ventricle is now over-distended. It has to and does accommodate, by the yielding of its elastic walls, more than the ordinary measure of blood. Thus, when the next systole commences, the chamber finds itself still more overweighted. At the end of the systolic effort there are again arrears of work. Occasional powerful, perhaps violent, contractions of the ventricle may relieve the chamber of its increasing accumulation, in response to the high internal pressure; but the succeeding systoles are again feeble, and the process of over-distention and overweighting is repeated. If the nervo-muscular energy continue insufficient,—that is, unless it recover or be restored,—the over-distention of the ventricle persists and passes into dilatation,—dilatation from incomplete emptying, *asystolic*.

The process of failure may affect any or all of the cardiac chambers, and may occur under a variety of circumstances. Not only pure hypertrophy, but compensatory dilatation with hypertrophy, as in aortic incompetence, may fail, and consecutive or "secondary" dilatation be established. This is, indeed, the chief way in which chronic valvular disease comes to be attended with symptoms and in which it directly or indirectly proves fatal.

Dilatation from failure also occurs independently of previous enlargement. If a heart of perfectly normal size be overtaxed whilst suffering from impairment of nutrition, as in anæmia, or from actual parietal disease, such as myocarditis or pericardial adhesion, it may either gradually or suddenly fail to complete its systolic work, and a condition of so-called *primary or idiopathic dilatation* be established. Such is the result of the associated increase of arterial pressure and granular degeneration of the myocardium which occur in scarlet fever. Nay, even when the walls of the heart are perfectly healthy, the parietal energy may prove insufficient to empty the chambers, if the resistance ahead be inordinately raised (*relative inadequacy*), particularly if the rise be sudden as well as extreme. This is what occurs in strain of the heart by violent muscular exertion, which in different cases induces passing embarrassment of the circulation from temporary over-distention of the chambers, or serious dilatation of various duration, or even sudden death. Fortunately, enlargement from strain is by no means common in the child. Lastly, these considerations enable us to understand how even moderate muscular effort may be sufficient to cause symptoms of distress when the heart is the seat of chronic valvular disease, however perfect the compensation and however sound the walls. A heart that is already handicapped by valvular lesion, and that accomplishes its work only by a continuous call on its reserve force, is readily overweighted by a comparatively slight effort, such as climbing stairs, or disturbed in its action by excitement. The sense of cardiac distress, dyspnœa, and palpitation, which arise under these circumstances, indicate temporary over-distention of the heart from parietal failure, a condition which will proceed to dilatation if it remains unrelieved.

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[1] In this discussion of the origin of dilatation, it is assumed that the ventricle normally empties itself in systole. Whether it does so completely or not does not affect the argument.

It will now be profitable to compare the three kinds of enlargement of the heart which have been separately analyzed, and to trace their mutual associations.

Hypertrophy of the heart is the result of a purely physiological process, the two essential elements of which are (1) increased muscular activity of the cardiac walls in response to increased demands for force; and (2) increased muscular growth consequent on increased activity. *Dilatation of the heart from overfilling* is the result of a purely mechanical process, the two essential elements of which are (1) over-distention of a cardiac chamber from overcharging, consequent on a valvular defect; and (2) increased capacity from continued stretching of its elastic walls. *Dilatation of the heart from failure and incomplete emptying,* whilst it also is the result of a purely mechanical process, implies a serious physiological inadequacy, depending as it does on work unaccomplished in consequence of a parietal weakness,—on inability of the muscular walls to complete the evacuation of the chamber, on inefficiency of the driving power to overcome completely the resistance ahead. Hypertrophy and dilatation from overfilling are methods of successfully meeting a difficulty or defect in the circulation by increased activity or elastic accommodation; dilatation from incomplete emptying is a process of yielding or breaking down in the face of a difficulty. The first two processes are indicative of relief, compensation, and safety: the third process is indicative of failure, disability, and danger.

The three conditions just described are variously associated in individual cases. Hypertrophy with dilatation from overfilling involves the left ventricle in every instance of aortic incompetence, and the right ventricle in the excessively rare lesion of pulmonary incompetence. The left auricle is more hypertrophied and less dilated in mitral obstruction, more dilated and less hypertrophied in mitral incompetence. In the latter disease the left ventricle is generally (not always) dilated and hypertrophied, in consequence of being overfilled by the excessive charge it receives from the enlarged auricle. The right ventricle is hypertrophied in mitral disease, the increased pressure within the left auricle exerting itself backward through the valveless circuit within the lungs until it falls upon the pulmonary valves. The same condition obtains in chronic pulmonary diseases attended with circulatory obstruction. But the hypertrophy of the right ventricle in these cases is rarely if ever pure: it is associated with dilatation from relative or actual failure. The right auricle is dilated from over-filling and moderately hypertrophied in tricuspid incompetence; it is found extremely dilated and also hypertrophied in tricuspid obstruction.

When adherent pericardium or other morbid conditions affect the whole cardiac wall, the disability involves all the chambers and induces general enlargement. Combinations of two or more kinds of valvular disease produce a great variety of forms of hypertrophy and dilatation, and the same is true of malformations of the heart. Finally, it must be added that, in every kind and case of cardiac enlargement proving fatal otherwise than suddenly, evidence will usually be found post mortem that dilatation from failure has been developed before death.

Effects on Other Organs.—When one of the cardiac chambers becomes progressively dilated from increasing failure of the propulsive force, the internal pressure begins to make itself felt backward upon the parts of the circulation behind the seat of over-distention (*Rückwirkung* of the Germans). This unfortunate process is most familiar in mitral disease, where it originates the symptoms of mechanical congestion and cardiac dropsy. If the left auricle fails, with imperfect emptying and dilatation, the pressure within the pulmonary circuit, already excessive, increases greatly, giving rise to still more dyspnœa, hæmoptysis, etc. The hypertrophied right ventricle next fails in the face of the increased resistance at the pulmonary valves, combined with the impaired nutrition of its wall; it undergoes secondary dilatation; and the tricuspid valve becomes relatively incompetent from dilatation of the opening and feebleness of the muscular structures in connection with it and with the valvular segments. The right auricle thus becomes over-distended and dilated; and, its own wall failing at the same time, in the presence of the increasing resistance to evacuation into the ventricle, the venæ cavæ and their branches are choked, and the radicles within the viscera and other structures that they drain suffer from mechanical congestion and its effects. Similarly, in failure of the left ventricle in aortic obstruction or Bright's disease, and in failing compensatory dilatation with hypertrophy in aortic incompetence, the mitral valve becomes relatively incompetent as the parietal weakness proceeds, and the same series of effects is set up as in primary mitral lesion. This is the origin of the morbid appearances found post mortem in the lungs, kidneys, and stomach, already mentioned, as well as of the dropsy and a long train of distressing symptoms to be presently described.

But, beyond this, the circulatory and visceral disturbances set up by secondary dilatation have an unfortunate effect upon the heart itself and the cardiac activity. The hepatic, gastric, and intestinal congestions and the catarrhs which they induce greatly interfere with digestion and sanguification in the child. Elimination by the bowels, kidneys, and skin is seriously diminished, and respiration is still further impaired. The coronary veins themselves share in the general mechanical congestion, seriously adding to the nutritive disturbances within the cardiac walls. Thus failure of the wall of the heart, like congenital malformation, comes ultimately to impoverish and poison its own blood-supply. In a word, a thoroughly vicious circle is established, the unfortunate effects of the disease of the heart recoiling on the organ itself.

Happily, this is not the end of every instance of failure of the heart. When compensation has been disturbed, either through actual weakness of the cardiac wall or excessive increase of the work that it has to accomplish, recovery may be effected by increasing the vigor of the myocardium or by diminishing the load that it has to drive, respectively. If this result be accomplished, whether by nature or by art, *compensation is said to be restored.* Most of our treatment in heart-disease is directed to this end, as we shall presently see. The heart, thus relieved and assisted, is again able to accomplish its work. The systole of the chambers becomes once more complete. There is no longer a residue at the end of the act, no accumulation, no arrears. The dilatation, as far as it originated in failure, has disappeared, and compensation, whether by pure hypertrophy or by compensatory dilatation with hypertrophy, is re-established, until, under the influence of the same or of other debilitating causes, it again breaks down.

Symptomatology.—The symptoms and physical signs associated with enlargement of the heart vary very widely, not only with the kind of enlargement present and the particular chambers involved, but also with the nature of the primary lesion,—valvular or otherwise,—to which the increase in size is but an adjunct.

Before proceeding to study this subject it is necessary to remember in what respects the *normal* clinical phenomena connected with the heart are peculiar in the child.

1. The *situation of the apex-beat* is, speaking generally, more to the left in children, lying beyond, in, or just within, the left vertical nipple-line, in the fourth or fifth interspace, according to the age and the growth of the diameters of the chest. More particularly, according to the observations of Von Starck,[1] the situation of the apex-beat is frequently indeterminate in the first years of life. It lies without the nipple-line in most children up to the fourth year; during the following years less and less frequently in this situation; after the thirteenth year practically not at all. It is found in the mammary line but seldom in the first year; more and more frequently so up to the seventh year; less often again after that age; but at fourteen may again be found there. Within the mammary line the apex-beat is never found up to the second year; seldom up to the seventh year;

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[1] Centralbl. f. Klin. Med., 1888, No. 34, p. 612.

from nine upward, in the majority of individuals; from thirteen upward, almost exclusively. In the fourth interspace the apex-beat lies almost exclusively during the first year; thereafter less and less often in that situation. The apex-beat is found in the fourth and fifth interspaces but seldom during the first two years of life; from the third to the sixth year often; thereafter again less often. In the fifth interspace the apex-beat is very seldom situated during the first two years; in the next years, more often; from seven onward, in the majority of subjects; after the age of thirteen, almost without exception there. The apex-beat is very seldom, indeed, to be found in the sixth intercostal space in children.[1]

2. The *cardiac impulse* is more widely visible and palpable in the child, the parietes of the chest being thin, soft, and elastic. This normal difference is exaggerated in children with cardiac disease, who are often peculiarly thin, or even wasted. In infants, on the contrary, the impulse may be very weak, or even imperceptible.

3. The *cardiac sounds* are peculiar in some children, possessing one or more of the following puerile features: (*a*) they are "deliberate"—*i.e.*, slow or hesitating—in character; (*b*) they are short, and therefore distinctly separated from each other,—*i.e.*, both periods of silence are unusually marked; (*c*) reduplication is more often present than in the adult; and (*d*) they betray occasional irregularity of rhythm.

4. The *radial pulse* is necessarily more frequent, and most of its characters, including regularity, are less definite, than in the adult.

Besides these points of physiological peculiarity, the practitioner will do well to bear in mind, when he is approaching the clinical investigation of a case of enlargement of the heart in a child (especially an infant), the great value of an acute eye and ear. He must be prepared to take in almost at a glance the child's general appearance, complexion, expression, attitude, and behavior and symptoms during examination, and to note, before the circulation is disturbed by crying, the appearances of the hands, cheeks, lips, neck, and praecordia.

HYPERTROPHY.—*Symptoms.*—It has been already shown, under the head of Pathology, that pure hypertrophy of the heart is a condition of perfect compensation. The subject of valvular disease under these circumstances suffers from no cardiac symptoms. If the valvular lesion have arisen in latent rheumatism in early childhood, not only may the patient make no complaint to direct attention to the heart, but the parents and the family practitioner may be unaware of the existence of cardiac disease. The palpitation, arterial throbbing, headache, and hemorrhages which occur in cardiac hypertrophy from chronic Bright's disease cannot be fairly attributed to the enlargement of the heart, but to the high tension which has set it up. The symptoms attending hypertrophy that is imperfect or breaking down are the symptoms of dilatation with failure, and as such will be presently described.

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[1] See also Keating and Edwards, Diseases of the Heart and Circulation in Infancy and Adolescence, 1888, p. 10; Gundobin, abstracted in Medical News, December 10, 1887, p. 683; and The Topographical Anatomy of the Child, by Johnson Symington, 1887, p. 64.

Physical Signs.—In a well-marked case of simple hypertrophy of the *left ventricle* in the child, inspection and palpation reveal a moderate degree of general bulging of the præcordia. The apex-beat is situated lower and more to the left than normal, probably in the fifth or sixth interspace, a variable distance without the left vertical nipple-line. The impulse is forcible, localized, and of well-developed thrusting quality. The præcordial dulness preserves very nearly the normal outline, in the form of a triangle, but with a somewhat wider base, running from the sternum to the apex-beat. (See Fig. 1.)

FIG. 1.

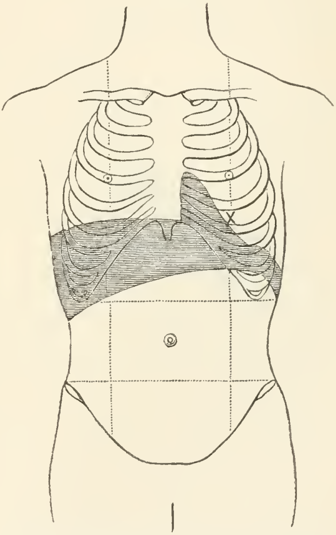

Diagram illustrating the cardiac (and hepatic) dulness and the situation of the apex-beat in a case of pure hypertrophy of the left ventricle, with aortic obstruction.

In the majority of cases there is an endocardial murmur, due to the primary valvular disease. Where this is absent, the first sound is of a dull, rather indeterminate but forcible character at the left apex, with absence of its ringing valvular element.

In simple hypertrophy of the *right* ventricle corresponding signs may be discovered over the chest and in the epigastrium.

Simple hypertrophy of the *auricles*, if it exist, cannot be determined physically.

COMPENSATORY DILATATION WITH HYPERTROPHY.—*Symptoms.*—Many of the subjects of this kind of enlargement of the heart are free from symptoms, whether the primary lesion be mitral or aortic incompetence. At the same time the child is thin, pallid, and probably undergrown; and it is found that severe exertion or excitement more readily induces cardiac distress than in pure hypertrophy or in the healthy subject. Whilst recovery is complete so far as the dynamic conditions of the wall are concerned, valvular incompetency—involving, as it does, abnormal distribution of the blood within the heart, the aorta, and the pulmonary circuit—necessitates certain symptoms. Children with mitral regurgitation, however perfectly compensated, suffer from the effects of comparative fulness of the pulmonary vessels, including dyspnœa, cough, and some duskiness of the extremities. Similarly the subjects of aortic incompetence always present in some degree the symptoms of general anæmia. When urgent symptoms, however, do appear in either case, they are due to failure of the heart, and their description strictly belongs to the next section.

Physical Signs.—In compensatory dilatation with hypertrophy of the *left ventricle,* the visible and palpable signs of enlargement and increased force are more marked than in simple hypertrophy. The child's præcordia bulge visibly. The apex-impulse is situated so low and so far to the left as to occupy the fifth, sixth, or seventh interspace, possibly in the anterior axillary line; it is extensive, powerful, and heaving, often with visible relapse of the soft parts over the spaces near or within the apex, and of the epigastrium. The area of percussion-dulness is correspondingly increased, and, as it extends farther transversely towards the left, the triangular outline presents an unnaturally wide base. (See Fig. 2.)

Fig. 2.

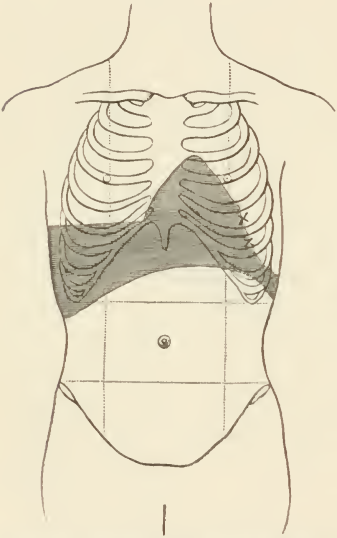

Diagram illustrating the cardiac (and hepatic) dulness and the situation of the apex-beat in a case of dilatation with hypertrophy of the left ventricle in aortic obstruction and incompetence.

Signs of direct compression of the left lung may sometimes be found at the posterior base. The auscultatory signs in cases of acquired heart-disease are always those of incompetence of the auriculo-ventricular or of the arterial valves, as the case may be, with their characteristic murmurs.

Dilatation from Failure.—*Symptoms.*—A description of the phenomena of failure of the cardiac walls will be most intelligible to the reader if it commence with the cases in which the process occurs in the course of compensatory enlargement and sets up the familiar symptoms of "heart-disease." It is when compensation breaks down that these—or, more correctly, most of these—make their appearance in the course of chronic disease of the valves or of the pericardium, their relative prominence varying with the seat of the primary disease and with the particular chamber that is failing.

The countenance of the child is anæmic, with a slightly livid tint, and expressive of suffering and anxiety. The body wastes, especially in the case of infants. The radial pulse fails in force and volume, whilst it rises in frequency and tends to become irregular; its tension and other characters are difficult or impossible to determine. In failure of the left ventricle, as in aortic disease, palpitation, pain, and other forms of præcordial distress, faintness, giddiness, and other effects of general anæmia are complained of. When the left auricle and the right ventricle fail in mitral disease, and the brunt of the trouble falls upon the lesser circulation, the urgent symptoms are dyspnœa, cough, hæmoptysis, and passive catarrh of the bronchi, with liability to acute pulmonary complications,—especially œdema, congestion, embolism, "apoplexy," and pneumonia; the cheeks present a dusky bluish-red flush; the eyes are suffused; the hands are livid and cold. Failure of the right heart also leads to that long series of disturbances in the venous circulation, due to mechanical congestion, which is clinically known as "cardiac dropsy." The chief of these are enlargement, pulsation, and tenderness of the liver, with functional disturbance and jaundice; derangements of the stomach and bowels; diminished volume and increased color and weight of the urine, with deposit of urates and the appearance of albumen; emotional depression; sopor or insomnia; epistaxis; and dropsy of the abdomen, chest, pericardium, and integuments.

This is a sketch of the phenomena attending a *severe* case of secondary cardiac dilatation,—of one which is much less common in children than in their seniors. It must not be understood that all these symptoms are present in every instance of failing heart. Indeed, in the great majority of cases the child comes under our care with but a few of the earlier symptoms,—namely, those directly referable to the heart itself and to the lungs.

The most constant of these is dyspnœa, about which the parents never fail to complain. Dropsy is less common, less pronounced, and more shifting. Pain and other subjective symptoms in connection with the heart, if not less felt, are certainly less complained of than by adult patients. Palpitation is frequently present. Acute pulmonary and pericardial complications are by no means uncommon in the young. Albuminuria is comparatively infrequent, even when the volume of urine is small and the liver greatly enlarged. Epistaxis is very common; and this, or premature menstruation, ought to excite the suspicion of cardiac disease.

Acute primary dilatation of the heart in febrile diseases is evidenced by distress and pallor (possibly with œdema) of the face; great feebleness, frequency, and irregularity of the pulse; lividity and coldness of the extremities; restlessness; sweating; accelerated, shallow respiration; and coolness of the breath.

Physical Signs.—In dilatation of the left ventricle from failure the impulse is feeble, indefinite, and diffused, below or beyond the left nipple, —*i.e.*, it loses force and definiteness of localization and character; or it may become entirely imperceptible. The area of percussion-dulness increases transversely, especially in dilatation of the right ventricle, when it passes to the right of the sternum and assumes the rude outline of a very flattened triangle with wide base and rounded angles. (See Fig. 3.) Along with these signs there occur in dilatation of the right side certain associated phenomena, such as marked disproportion in strength between the epigastric impulse and the radial pulse, loudness of the second pulmonary sound, and fulness of the cervical veins. In emphysema the dulness is obliterated by the pulmonary enlargement, and the heart and its signs are dislocated downward into the epigastrium. The auscultatory signs are very striking. In valvular disease murmurs that existed before the supervention of the failure lose their quality and intensity, and may disappear from very feebleness. On the contrary, in primary dilatation the relaxation of the myocardium, the dilatation of the auriculo-ventricular orifices, and the consequent ("relative") incompetence of the mitral or the tricuspid valves, may be attended by the development of a systolic apex-murmur, probably variable and temporary. These two phenomena may be combined in failing right ventricle in mitral stenosis,—the presystolic murmur disappearing and a tricuspid regurgitant murmur being developed.

Fig. 3.

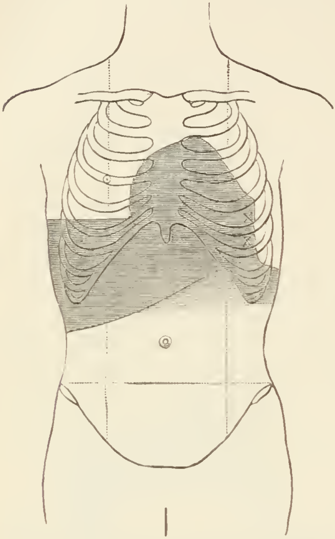

Diagram illustrating the cardiac (and hepatic) dulness and the situation of the apex-beat in dilatation of the heart from failure. Both left and right sides are involved. Primary lesions, double mitral disease, and adherent pericardium.

Where a valvular murmur does not exist, or has disappeared, the first sound at the apex assumes a clear, sharp, short, "flapping," valvular character, which suggests loss of the muscular element; in the acute primary dilatation of fever it may entirely disappear.

Diagnosis.—Diagnosis has to be carried out in connection with enlargement of the heart as regards four important points,—viz., (1) the diagnosis of cardiac enlargement from conditions that simulate it, (2) the diagnosis of the three kinds of enlargement from one another, (3) the diagnosis of the seat of the enlargement, and (4) the diagnosis of the cause of the enlargement.

(1) *Diagnosis of cardiac enlargement from conditions that simulate it.*—The pigeon-breast of rickets will not be mistaken for bulging of the præcordia from enlargement of the heart if the exact situation and outline of the prominent area be regarded: pigeon-breast is a symmetrical fulness; in cardiac enlargement the bulging corresponds with the position of the heart in the chest, lying mainly to the left side. Displacement of the heart and apex-beat is readily diagnosed from enlargement by determining the presence of the cause of dislocation and the absence of a cause of enlarge-

ment, as well as by a careful study of the area of præcordial dulness, which is bodily transferred to a new position in displacement, but only increased in its limits in enlargement. Abnormal pulsation in connection with aneurism of the aorta or with malignant growths must not be confounded with the cardiac impulse of enlargement. Pericardial effusion is differentiated by the shape of the increased area of dulness, and by the characters of the impulse, the sounds, and the adventitious signs.

(2) *Diagnosis of the three kinds of enlargement from one another.*—This ought to be readily indicated by the presence of the symptoms in dilatation from failure, such as palpitation, pain, and dropsy, which are practically absent in compensatory enlargement. As regards physical signs, the following points specially indicate dilatation from failure: the character of the impulse, the shape of the præcordial dulness, the characters of the sounds, and the relation of the extent of dulness to the strength and definiteness of the impulse and to the sounds.

Pure hypertrophy and compensatory dilatation with hypertrophy are distinguished from each other, say in the case of the left ventricle, by physical signs, the impulse being more extensive, more powerfully heaving, and situated behind as well as below the left nipple in dilatation with hypertrophy, whilst the præcordial dulness is specially increased transversely.

(3) *Diagnosis of the seat of the enlargement.*—This depends on an intelligent consideration of the symptoms and signs already described, and of the seat and nature of the valvular disease or other primary lesion.

(4) *Diagnosis of the cause of the enlargement.*—When the primary lesion is situated in the valves, wall, or coverings of the heart, it is, as a rule, readily discovered by physical examination and study of the patient's history; and extrinsic causes, such as disease of the lungs or kidneys, should also be easily recognized.

The diagnosis of the cause of dilatation from failure is less easy, but must always be attempted. In every individual instance that comes before him, the practitioner should ask himself, What has happened, that this patient who was previously free from symptoms is now suffering from dyspnœa, palpitation, and dropsy?—and he ought not to be satisfied until he has exhausted every means of answering the question.

Prognosis.—The prognosis of cardiac enlargement in the child is a question of compensation. Whatever circumstances promote compensation will improve the prognosis; whatever circumstances promote dilatation from failure must be regarded unfavorably.

1. In *simple hypertrophy* and in *compensatory dilatation with hypertrophy* the prognostic question that arises is, Will the compensation be maintained, or will it fail and secondary dilatation take place? This question can be answered only after a careful estimate of all the circumstances in which the child lives.

On the one hand, the conditions which we found in the section on pathology to be required for the establishment and maintenance of compensation are peculiarly present in young subjects. Growth in size and increase of vigor are active processes in children; the coronary vessels are sound; the nervous influences are healthy; the elasticity of the cardiac walls and blood-vessels is perfect; the lungs and the eliminating organs—indeed, all the viscera—are comparatively unimpaired. The weight of the heart normally doubles itself at puberty,—an indication of a large reserve force. Speaking of compensation in the juvenile heart, Dr. Jules Simon truly says, "Son rôle commence au lieu de finir."[1] As a matter of fact, conservative enlargement occurs with exceptional completeness and rapidity in the early years of life, and a child may be expected to remain practically free from symptoms as long as the circumstances of life are favorable.

On the other hand, the child, like the adult, is constantly threatened by conditions which tend to undo compensation. *Dyspepsia*, from poverty in the case of the poor, from over-feeding and coddling in the rich, and *anæmia* in connection with overgrowth and development—especially at critical ages and in anticipation of puberty—or from hemorrhage, will impoverish the blood, and thus lower the nutrition of the myocardium. Local anæmia from degeneration of the coronary vessels may fortunately be discounted prognostically in the child, but pericardial adhesions may tend to set up serious parietal debility. *Nervous* influences, so fruitful a source of broken compensation in the adult, may also be comparatively disregarded in young subjects: first, because far less numerous, and, secondly, because children can be spared so many shocks and strains of this kind which their elders must suffer. A class of causes of cardiac failure that have to be specially anticipated in the juvenile subject of compensated enlargement are *intercurrent diseases*. Rheumatism is peculiarly to be dreaded. If a child with chronic valvular disease of old rheumatic origin continues to suffer from rheumatism, however slight, or if better-declared attacks occur at short intervals, the heart is in constant danger of failure, with cardiac suffering, occasional pericarditis and myocarditis, possibly extension of the original endocardial lesion, and sometimes acute pulmonary and pleuritic complications. From the tenth or twelfth year till puberty may be expected to be the most unfavorable age in this respect. Intercurrent bronchitis and pneumonia, as well as the acute specific fevers, should also give rise in the mind of the practitioner to anxiety in young subjects with damaged but compensated hearts. The occurrence of chorea must be similarly regarded. *Muscular overwork* and its effects on the arteries and heart may be almost neglected as unfavorable circumstances when we are forecasting the maintenance of compensation in a child. Whilst adults *must* work, and thus rarely escape the effects of overtaxing their myocardium, children *need not* over-exert themselves, for trying games, which are the chief cause of muscular strain in young cardiac subjects, will be interdicted. In the same way, most children may be confidently expected to escape those *toxic* causes of cardiac failure so frequent in the adult,—alcohol, tobacco, tea, gout, and syphilis.

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[1] Revue Gén. de Clin. et de Thérap., Dec. 29, 1887, p. 714.

It will be gathered from these remarks that the prognosis of the maintenance of compensatory enlargement of the heart in a child depends greatly on the social position of the patient. If he belongs to poor and ignorant parents, his future is unfavorable, not only because he will be exposed to one or all of the unfavorable influences just considered, but also because he will not have the advantages which the well-to-do child enjoys of *early*, close, and prolonged medical attendance in acute diseases, of periodical examination of the heart, to estimate its vigor and to detect any slowly progressive valvular change, and of constant supervision in his physical and intellectual education.

2. When *dilatation from failure* has set in, the prognostic question that arises is, Can the compensation be restored? The answer to this question depends upon which of the causes of failure is at work in the case before us.

The prognosis will be *favorable* when the cause can be discovered and removed. Thus, in failure from impaired general nutrition, the blood will be restored with comparative ease in the child whose digestive and hæmopoietic organs are sound, in whom mechanical congestion has been of short duration and the consequent fibroid change still insignificant. Similarly, an encouraging forecast may be given in failure from moderate muscular overwork, where the child can be carefully treated and watched at home or in hospital. On the contrary, the prognosis will be *unfavorable* when the cause of the dilatation is irremovable,—for instance, in acute intercurrent diseases, rheumatism, chorea, acute specific fevers, and acute pulmonary disease. In many instances, again, the prognosis will prove to be obscure and uncertain because the causes of failure are undiscoverable or variable. This is the case when depressing nervous influences are at work, and also when there is increase of the original lesion in consequence of recent endocarditis or pericarditis.

The appearance of the more advanced symptoms of dilatation, including dropsy and albuminuria, is very unfavorable, indicating, as it does, a much more serious rupture of compensation than it would in the adult. *Per contra*, the prognosis becomes more favorable the longer compensation is maintained.

Children seldom die directly of cardiac disease, but indirectly of acute complications. Sudden death from heart-disease, whilst it does occur, is uncommon in the child.

Treatment.—Whilst it is not a substantive disease in the proper sense of the word, cardiac enlargement is the chief key to treatment in all organic affections of the heart. Speaking generally, compensatory enlargement is to be promoted and maintained, failure to be prevented or removed, whatever their cause. Little can be done for valvulitis or for adherent pericardium; much may be done to favor the hypertrophy which is a natural remedy of the dynamical disturbance, or to recover the heart from the condition of debility and dilatation into which it may have fallen.

SIMPLE HYPERTROPHY, AND HYPERTROPHY WITH COMPENSATORY DILATATION.—The question of the proper treatment of these conditions arises at two periods in the course of disease of the heart in children. First, after convalescence from acute endocarditis or pericarditis we have to ask ourselves anxiously, What measures will encourage the development of compensatory hypertrophy or dilatation? Secondly, when compensation has been established, and the child is free from symptoms, we have to consider how the conservative enlargement is to be maintained.

The treatment that has to be followed at these two periods is practically the same. It is partly of a positive kind,—*i.e.*, it consists in actively carrying out certain rules of life and methods of therapeusis,—and partly is negative,—*i.e.*, it includes the faithful avoidance of certain unfavorable circumstances.

1. The first end to be secured is a sufficient supply of *healthy blood* in the coronary vessels. On this subject it is unnecessary for us to enter into details: abundance of pure blood is the product of perfect hygiene. Nevertheless we must confess that in young cardiac subjects this end is very difficult of attainment, whether among the poor, from obvious reasons, or among those in happier circumstances, where there is a constant temptation to coddle and over-feed the delicate child, particularly as he is likely to be thin. As it must never be forgotten that active excretion is as necessary for a healthy blood-state as abundance of food, the bowels, urine, and skin must be faithfully watched by the mother, and occasionally specially stimulated. Warm woollen clothing is essential. Iron or iron and arsenic are indicated in these subjects.

2. It is all-important that the child should be subjected to *wholesome nervous influences.* This part of the general treatment of cardiac disease practically resolves itself into a question of education, which will have to be carefully considered in each instance, and is confessedly difficult. The practitioner must insist on the perfect recovery and maintenance of health, so far as these are possible, before he allows the child to return to school; and it may not be until months after an attack of rheumatic endocarditis that he can safely do so. When lessons are resumed, the parents or teachers must be on the outlook for headaches, insomnia, sleep-talking, twitching, irritability, excitability of manner, the display of precociousness, or the symptoms of chorea; and they must act promptly if any of these arise.

3. Along with the subject of schooling that of muscular rest, exercise, play, and amusements has to be settled by the medical attendant. When the period of rest after cardiac inflammation is ended, he will have to say definitely whether cricket and foot-ball are to be allowed or not, and to speak unhesitatingly as to other games and athletics. This calls for the employment of great judgment. One obvious rule to follow is to forbid all matches, whilst more or less "stupid" games of cricket and tennis may be

permitted. Foot-ball and paper-chases are to be entirely forbidden: there is too much muscular strain and prolonged and increasing excitement in these games for delicate chests. Tricycling may also have to be interdicted, unless the country be level and the boy's ambition moderate. Bathing must be interrupted for a time, whether in the sea, river, or swimming-bath. In this connection it may be mentioned that the sea-side as a whole is less suitable for cardiac subjects than bracing inland places, where they will have wholesome walking and be surrounded by abundance of reviving, strengthening, and yet soothing influences, with none of the exciting effects of the coast. For the first few weeks or months after complete recovery from acute heart-disease, it is a good plan to send the child to a quiet country place, there to spend an out-door life with his companions,—always under faithful observation. When compensation is completely established, muscular exercise should be ordered, the amount and the kind depending upon the nature of the primary lesion. Girls may be treated much like boys. Dancing will have to be forbidden them for a time, to be gradually resumed in the gentlest form under perfectly non-exciting circumstances.

4. In children who are the subjects of chronic cardiac disease with compensation, it is of the first importance to prevent the *incidence of acute diseases,* and, if such do occur, to sustain the heart during the attack and the subsequent convalescence. In scarlet fever and measles the heart, pericardium, and lungs must be watched with unusual care, and immediate attention paid to symptoms indicative of acute dilatation, the timely use of purgatives and diaphoretics being indicated in scarlet fever with acute renal congestion.[1] Alcoholic stimulants, ether, ammonia, strychnine, and the digitalis group of drugs will be called for. Equally important is it to guard the child against primary bronchitis and pneumonia.

But the intercurrent disease which has to be specially prevented is rheumatism. The practitioner must not forget that this may be insidious, perhaps latent, in its manifestations, and, once established, may haunt the joints and heart for months; that, however slight its effects in the joints, it will quickly and seriously undo compensation unless it be immediately combated. The measures best calculated to prevent rheumatism are mainly those already enumerated under the heads of the blood and exercise. If articular pain and pyrexia make their appearance, our endeavor must be to cut short the rheumatic attack by appropriate treatment. The greatest patience may be demanded of the practitioner in protracted cases of this kind. Repeated relapses of rheumatism week after week may discourage him, and threaten to destroy the confidence of the parents. These difficulties are best overcome by being met half-way: he must show that he is not unprepared for the irregular indefinite reappearance of the rheumatism and its attendant cardiac, pericardiac, and possibly pulmonary complications. Treatment, of the strictest anti-rheumatic kind, must not be shifty and faltering, but consistent and continuous. Absolute rest of mind and body, the best procurable nursing, rigidly simple fluid diet, and daily evacuation of the bowels, added to the employment of salicylates or other appropriate medicinal remedies, will be the surest means of carrying a child safely through such an attack. In no class of cases does good treatment consist more distinctly in *management*,—of the parents as well as of the child. When the pains and pyrexia have disappeared, nothing less than the continuance of rest and low diet (so difficult to insist on in these subjects) for several weeks will prevent a fresh relapse and insure satisfactory convalescence.

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[1] Goodhart, loc. cit.

Intercurrent chorea is best prevented by faithful attention to the points already touched on under the heads of rheumatism, education, and the nervous system.

DILATATION FROM FAILURE.—When failure of compensation has taken place, and the child is suffering from cardiac symptoms, treatment of another kind is called for.

1. *Treatment of the Cause.*—At this stage also, as in compensation, the first consideration in treatment is the avoidance of routine. Unless the symptoms are urgent, we must not at once fly for help to ether, digitalis, ammonia, or alcohol. Rational therapeusis begins with attention to the cause of the morbid state calling for remedy; and we have seen that cardiac failure is not a substantive disease, but an effect of many possible causes. Our first duty is to discover the origin of the break-down and remove it if possible. Here again, as in prognosis, we appreciate the importance of having searched out the cause of the secondary dilatation. Knowing this, not only may we be able to attack the morbid state directly, but our choice of remedial measures becomes more extensive and their application more easy. We are not confined to a few habitual "cardiac" drugs and methods, but can turn to account a range of hygienic and therapeutic measures as extensive as the causes of failure which they are intended to overcome.

(1) In many instances of secondary dilatation of the heart in children, including most of those met with in hospital and dispensary practice, compensation may be restored by little more than feeding and rest alone. These are the cases in which poverty, starvation, and anæmia have been traced as the sequence of events in the development of *impaired general nutrition* and consequent failure of the heart. This class of patients have to be temporarily rescued from a life which is practically incompatible with perfect compensation, and sent into the wards, where a few weeks of rest, warmth, and careful feeding, accompanied after some days with hæmatinics, will speedily dispel the cardiac symptoms. In other instances the same line of treatment must be pursued at the child's home.

(2) By way of contrast it is well to mention here the proper treatment of the opposite class of cases, in which misapplied care and *over-feeding* have undone the healthy nutrition of the heart. A spare and simple diet and wholesome daily exercise must be insisted on. More particularly the food must be anti-rheumatic,—containing the minimum of sugar, cooked fats,

and red meats. The bowels must be opened with active purgatives, such as jalap, scammony, calomel, or gray powder in combination with rhubarb and soda. When the worst symptoms have disappeared, regular exercise in the form of walking on the level must be commenced.

(3) When *muscular exertion* is discovered to be the cause of the failure of compensation,—*e.g.*, in boys at school,—treatment is to be conducted on the principles laid down under the head of exercise. The practitioner may at first have to order entire rest in bed; or he may act more judiciously by simply forbidding exertion and every kind of game that involves it. Above all, he will find that *time* is the chief element of cure in these cases: the effects of over-exertion may require many months for their undoing.

(4) In speaking of the maintenance of compensation we have already discussed the treatment of failure of the heart from *acute intercurrent disease.* It is unnecessary to return to this subject.

2. *Treatment of the Effects.*—When the cause of dilatation from failure cannot be discovered, or cannot be removed, we must proceed to treat the effects,—the dyspnœa, palpitation, and dropsy; and the same principle must guide us when the symptoms from which the child is suffering are too urgent or too advanced to justify the delay that often attends the treatment of the cause. Immediate relief of distress and danger by every means in our power is then our duty, as well as the most rational and successful system of treatment. The reader will find in the article on chronic valvulitis a full and practical account of this part of the treatment of disease of the heart. In the present article it will suffice to indicate the general principles that must guide us when we attempt to restore compensation.

(1) *Increase of the Cardiac Force.*—In acute primary dilatation, as it occurs in fever, and in most instances of failure in cardiac enlargement, the first and easiest method of relief is stimulation of the heart. The cardiac action may be increased in force, and rendered more effective by altering the frequency and securing better rhythm. The most rapid and powerful cardiac stimulants are subcutaneous injections of ether and of the one-per-cent. solution of hydrochlorate of strychnine. Combinations of equal parts of spirit of ether and sal volatile in water, frequently repeated, are most effective internal remedies in urgent cases, both cardiac relief and free diuresis often following. Alcohol in the form of spirits is invaluable, being not only powerful but also always available; and there are few instances in which it is not to be given, combined with water or with liquid food, such as eggs, milk, broths, and jellies. When the condition is less urgent, we usually prescribe a remedy belonging to the great group of cardio-vascular tonics,—digitalis, strophanthus, squill, senega, or convallaria, or, it may be, caffeine. While there can be no question that digitalis is indicated in all valvular affections where compensation has not been effected,[1] it is equally true that there are many details with respect to the relative value, selection,

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[1] M. H. Huchard, Quand et comment doit-on préscrire le Digitale? Paris, 1888.

and employment of these remedies that demand faithful consideration in each case. These are discussed in Dr. Sansom's article. Digitalis and its allies do not act simply on the heart by increasing the systolic force. They also lengthen diastole (reduce the frequency), raise the general arterial pressure directly, and after a time relax the renal vessels, thus inducing free diuresis and greatly relieving the circulation. Strychnine may be combined with great advantage. As the condition improves, iron should be cautiously added to the mixture.

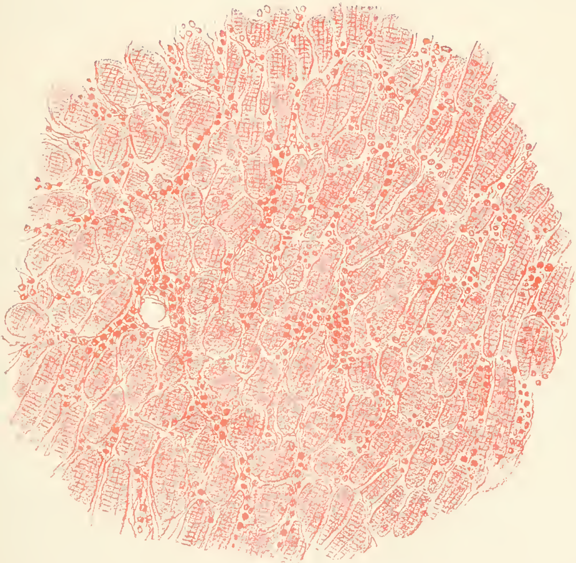
Various methods of reflex stimulation may be combined with medication when the symptoms are urgent, including sinapisms to the præcordia and calves, ammonia to the nose, and the admission of fresh cold air to the surface by ventilation and the use of the fan.

It is essential at the same time that food of a kind that shall supply nervo-muscular energy quickly and abundantly be perseveringly administered.

(2) *Relief of Over-Distention and Mechanical Congestion.*—Along with increase of force, we must attempt to afford the heart relief from what may be described as the burden of arrears of work,—the accumulation of undischarged blood within its chambers, the passive hyperæmia of the viscera, the serous effusions and dropsy which are sapping nutrition. There are three principal ways of fulfilling this indication:

(*a*) *Direct abstraction of blood.* In urgent cases of failure of the right ventricle, as in pulmonary and mitral disease, there is no means of relief so speedy and sure as venesection. This is now very rarely practised on children; and the same may be said of cupping, dry and wet. Leeching over the chest gives remarkable relief, however small may be the quantity of blood removed. (*b*) *Paracentesis abdominis* for cardiac dropsy is very successful in children. *Puncturing* of the legs or feet is best avoided in the young. These operations must always be practised with care and under strict antiseptic arrangements. (*c*) *Drainage of the engorged viscera*, by means of diuretics, hydragogue purgatives, and expectorants, is a method of removing the incubus of circulatory arrears which can always be practised. The use of cardio-vascular diuretics has been already referred to. With these may be combined direct renal stimulants, such as spiritus ætheris nitrosi and spiritus juniperi. Mercurial purgation will materially assist these drugs. Of hydragogue purgatives the best is compound jalap powder. Squill is the typical expectorant in cardiac failure, in combination with other cardiac stimulants and carbonate of ammonium.

(3) *Reduction of the Load to be driven.*—Rest in bed is the most obvious means of diminishing the work to be done by the heart when systole is not completed. The measures recommended in the last section have directly or indirectly this effect,—reducing the volume of blood by hydragogue action upon the bowels or kidneys, and removing the obstacles to the circulation presented by ascites and œdema. Direct arterial dilators have a much more rapid, if more transient, effect in the same direction, such as



Lympho-Sarcoma of the Myocardium, originating in the Mediastinum.—The growth consists of small round cells infiltrating the intermuscular lymphatic spaces. (Drawn by Mr. Collings from a preparation by Dr. Mott.)

nitrite of amyl, nitro-glycerin, nitrite of sodium, and spiritus ætheris nitrosi. These are severally used according to the urgency of the case, and are specially valuable in aortic incompetence and failure in Bright's disease.

(4) *Relief of Distress.*—In every instance we must try to alleviate pain, allay palpitation and anxiety, and therewith restore digestion and nutrition. This portion of the treatment of failing heart demands extensive general knowledge of therapeutical means, and its combination with that adapted to fulfil the primary indications severely taxes the skill of the practitioner. The measures to be used are described in the article on valvular disease, under "Sedatives."

NEW GROWTHS AND PARASITES.

New growths of the heart in children belong to the curiosities of medical literature. Tumors involving the myocardium are rare at all ages, and when they do occur are almost confined to the period of adult life. Of twenty-eight cases of malignant growths in the cardiac walls, collected by my friend Dr. Quain, in his hitherto unpublished Lumleian Lectures on diseases of the walls of the heart, which he has kindly allowed me to consult, I find that only two occurred in children,—at the ages of three days and twelve years respectively.

The kinds of new growth that have been recorded include myoma, fibroma, lymphadenoma, lipoma, carcinoma, and sarcoma. Miliary tubercles and scrofulous masses are more frequently met with when carefully searched for. Syphiloma of the heart is described in the article on myocarditis.

Pathology.—There is little that calls for special remark in the structure, etiological relations, or nature of new growths in the infantile or puerile heart. Perhaps the most interesting of all in the present connection is the myoma, or muscular-tissue tumor, inasmuch as it appears to be a congenital disease. Myoma of the heart, as described by Virchow and others, takes the form of multiple growths, some of them as large as a cherry or even a pigeon's egg, scattered throughout the walls of the different chambers. They are recognized as firmer, paler masses, apparently embedded in the myocardium, but really in complete continuity with its fibres, or making their appearance on both the endocardial and pericardial surfaces. Structurally they consist of striated muscular tissue, in very loose bundles, traversing large, irregular, hollow spaces, so that the whole texture of the growth is peculiarly cavernous. The accompanying plate illustrates a remarkable case of malignant lympho-sarcoma invading the heart in a young subject.

Echinococcus is the only parasite recorded in the heart of the child, and the instances of its occurrence are exceedingly few.

Symptoms, Diagnosis, Prognosis, and Treatment.—New growths and parasites involving the heart do not produce sufficiently defined symptoms during life to justify a diagnosis, nor, as a matter of fact, to excite a suspicion of their presence. Cardiac phenomena supervening in the course of lymphomatous, tuberculous, or malignant disease in a child would naturally suggest involvement of the myocardium; but similar symptoms might be even more reasonably referred to involvement of the mediastinal glands or the pericardium. The prognosis is hopeless, and the treatment is palliative only.

CHRONIC ENDOCARDITIS.—VALVULAR DISEASE.

By ARTHUR ERNEST SANSOM, M.D., F.R.C.P.

CHRONIC VALVULAR DISEASE.

Definition.—An abnormal condition of any portion of the valvular apparatus of the heart the result of previous disease.

Pathological Anatomy.—The most common morbid appearance in cases of valvular imperfection in children is a thickening of the mitral valve and the adjacent endocardium; the endocardium of the left auricle and ventricle is sometimes similarly affected; exceptionally a like thickening implicates the aortic valves; the change, however, may not be sufficient to impair their efficiency. Like thickening of the endocardium of the right chambers of the heart may occur especially in fœtal and early infant life. The most frequent result of this change in the endocardium and valve-structure is *mitral insufficiency,* so that the valve fails to close the orifice perfectly during ventricular systole. The result next in point of frequency is a welding and thickening of the mitral curtains in such a manner as to impose an obstacle between auricle and ventricle,—*mitral stenosis.* The thickening is often considerable, and the substance may assume the appearance of cartilage; exceptionally it may be hardened by infiltration with calcareous salts.

Disease of the right chambers may induce *tricuspid incompetence,* and hence regurgitation; but *tricuspid stenosis* in the period of child-life has not been recorded; with comparative rarity the aortic valves are thickened and puckered so as to render them incompetent, or, still more rarely, so as to produce *aortic stenosis.*

The diseased endocardium may be the seat of excrescences or vegetations. The most frequent situation of these is the auricular portion of the mitral curtains, but they are found also on the endocardium of the auricle or the ventricle and on the tendinous cords. The aortic is next in point of frequency to the mitral the seat of vegetations, the tricuspid comes next, and lastly the pulmonic. In some instances all the cardiac valves are found to have vegetations attached to them. In some examples the attachment is very slight, so that the excrescences—which consist of fibrin—are easily removed; in others they cannot be rubbed off,—they are warty outgrowths from the endocardium itself. Occasionally, although very rarely in young children, the endocardium is eroded from ulcerative endocarditis.

Concurrent Affections.—Of these the most frequently observed is pericarditis. The pericardium is often found adherent, and sometimes fibrous bands extend between the muscular fibrillæ. In cases where there is such adhesion the heart is often found greatly enlarged and its chambers hypertrophied, or dilated as well as hypertrophied. It is common also to find concomitant evidences of pleurisy. Of associations which are also probably predisposing causes, *malformations* are chiefly to be mentioned. In cases of malformation of the heart not only thickening of the valves but also vegetations are frequently observed.

Condition of the Muscular Tissue of the Heart.—The chief variation from the normal in regard to the muscle of the heart, especially of the ventricles, is *hypertrophy.*

If one compares the conditions with those observed in case of the adult, one can scarcely fail to be struck with the fact that this hypertrophy is disproportionate. It is to be remembered, however, that in the child in a largely preponderating number of cases pericardial adhesion is a concurring sign; it is most probable that such adhesion, implying the invasion of the muscular tissue with fibrous ingrowths and thus contributing with the valvular imperfection in calling upon the heart for increased effort to overcome its difficulties, is a cause of such disproportionate hypertrophy. In some cases dilatation of the chambers, ventricles or auricles, is manifest, and in a minority fatty degeneration is found.

Etiology.—In a large majority of cases chronic valvular disease in infants and children is the result of rheumatic endocarditis. It would appear that in rheumatism the endocardium is more vulnerable in the child than in the adult. The writer has found that of the cases of acute and subacute rheumatism treated at a children's hospital where patients were not admitted after twelve years of age, valvular disease at the time of the patient's leaving the hospital was manifest in from fifty to sixty per cent. This may not be a higher proportion than obtains in the adult, but it is shown that endocarditis having all the essential characters of the rheumatic may develop in children who present no articular signs of acute or subacute rheumatism, who may manifest slight or transient pains, or who may even present no sign of rheumatism whatever. Rheumatic endocarditis giving rise to chronic valvular disease may arise and progress with no definite sign to mark its onset and course. Scarlatina and measles are sometimes attended with or followed by endocarditis which results in chronic valvular disease: in these cases the pathological conditions are indistinguishable from those of the rheumatic form. The most frequent result of rheumatic endocarditis is a thickening of the valve or a retraction of its curtains, which brings about its imperfect closure at the time of ventricular systole and permits regurgitation into the left auricle. It is evident, however, that in a minority of cases a like form of endocarditis may induce fusion of the mitral curtains at their junction, and may cause mitral stenosis. Concerning the vegetations which form in many cases on the surface of the diseased endocardium, the evidence seems to point to the conclusion that in the majority of cases no other cause is at work for their manifestation than that rheumatic process which induces the inflammatory change and subsequent fibrous transformation of the membrane. In some instances there may be papillomatous outgrowths, in others merely adhesion of fibrin to little cups or depressions where the inflammatory process has deprived the membrane of its normal smoothness. In cases, however, of some forms of vegetation, there is good reason to believe that a septic cause is in operation either concurrently with or subsequently to the rheumatic; this is probable where there are exuberant vegetations, for it is distinctly proved that micro-organisms may infiltrate the endocardium and such vegetations, even though there may be no distinct and obvious losses of tissue. Therefore in the strict sense there may be a septic, even though there may not be an ulcerative, endocarditis.

It is probable that *violence* to the valves may also be a factor in the production of the endocarditis which results in chronic lesions in children. The writer is of opinion that *traumatic endocarditis* is more common than is generally supposed. In certain cases of chorea there is no sign of rheumatism nor of proclivity thereto, but a distinct history of sudden fright (a section of the cases of chorea which comprises nearly half the total number): it appears very probable that endocarditis when observed—a form of the disease characterized by papillomatous outgrowths fringing the margins of the mitral or aortic valves near their lines of closure—is due to the sudden violence done to the delicate structure of the endocardium, especially delicate in the young child, induced by the violent palpitation of the heart consequent upon the terror.

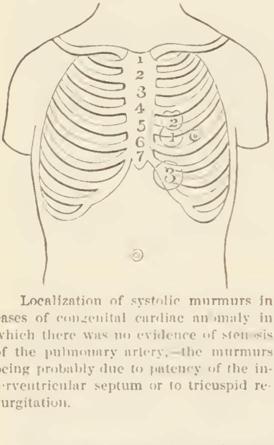
MITRAL INADEQUACY.

Definition.—A pathological condition of the mitral orifice inducing mitral regurgitation,—*i.e.*, reflux in systole into the left auricle.

Pathological Causes.—In the child the condition of imperfect closure of the mitral orifice at the time of ventricular systole giving rise to regurgitation of blood into the left auricle may be brought about by (*a*) structural alteration of the mitral valve or its attachments, (*b*) dilatation of the ventricle, so that the curtains of the valve fail to coapt, (*c*) a change in the muscle of the left ventricle whereby it fails sufficiently to approximate the segments of the valve during systole. This latter may be from inflammatory change (myocarditis) or from fatty degeneration of the muscular fibrillæ.

Diagnosis.—It may be affirmed that a defined murmur heard at the time of the ventricular systole over the position of the apex of the heart where the left ventricle strikes the wall of the chest, in some cases conducted towards the left axilla, and in some heard below the angle of the left scapula, is indicative of mitral regurgitation. An apparent exception is possible in the case of pericarditis when a murmur indistinguishable from the systolic murmur of mitral regurgitation may be heard, and yet the post-mortem examination may demonstrate the absence of valvular imperfections; but even in this case it is probable that from the myocarditis which accompanies the pericarditis the muscular power of the ventricle is impaired, and regurgitation into the auricle results from the imperfect approximation of the mitral curtains owing to the enfeeblement of the ventricle and the papillary muscles. In a large majority of instances a persistent systolic murmur at the apex indicates structural alteration of the valve or its attachments; but there are notable exceptions. One has just been noted in the condition of myocarditis which accompanies pericarditis and which may also accompany other febrile affections. Although long persistent, such murmurs, on the restoration of the strength of the ventricle, will disappear. In other cases the systolic murmur may be due to dilatation of the ventricle without any disease of the valves. In others, though with comparative rarity, it may be due to fatty degeneration of the ventricle. Fatty degeneration may be suspected when in addition to the signs to be noted there is a very marked *anæmia.*

Fig. 1.



Localization of systolic murmurs in cases of congenital cardiac anomaly in which there was no evidence of stenosis of the pulmonary artery,—the murmurs being probably due to patency of the interventricular septum or to tricuspid regurgitation.

A possible difficulty of diagnosis may occur in instances of congenital disease or anomaly. In such cases systolic murmurs may be heard, but the maximum intensity of these will be found to be to the right of the position of the apex, and they may be indicative of perforate interventricular septum or of tricuspid regurgitation the result of intra-uterine endocarditis. The accompanying diagram (Fig. 1) indicates the position of such murmurs in cases observed by the writer.

In these cases the existence of cyanosis or venous turgescence is an aid to the diagnosis, and the probability of the affection is greater the younger the infant. Such probability excluded, the differential diagnosis of the mitral regurgitation due to valvular disease from that due to the other causes mentioned may be difficult. The following rules may be useful:

1. A murmur of mitral regurgitation in a child manifesting any signs, however slight, of past or present rheumatism indicates most probably an imperfection of the valve the result of endocarditis.

2. If such a murmur be left after an acute attack of pericarditis, the diagnosis is doubtful; there is a possibility, though not a probability, that it may pass away and that no organic change may result. In the case of an apex systolic murmur developing in definite relation with an acute

pyrexia uncomplicated by rheumatism, the probability of the murmur being transient is very great.

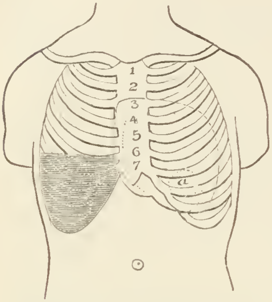
In the case of the manifestation of very marked anæmia in a child the subject of a mitral regurgitant murmur, fatty degeneration of the heart-muscle may be suspected.

Clinical History, Progress, and Symptomatology.—I. *Chronic Mitral Inadequacy the Result of Rheumatic Endocarditis.*—In a considerable number of cases the rise and progress of the form of endocarditis which results in the production of imperfection of the mitral valve and consequent mitral regurgitation may be traced in definite relation with rheumatism. As a general rule, it may be said that of the children admitted into hospital for acute or subacute rheumatism fifty to sixty per cent. are discharged with valvular disease, the most frequent form of which is mitral insufficiency. This, however, by no means represents the whole truth as to the influence of rheumatism as a factor of the endocarditis giving rise to the valvular imperfection, for it is abundantly proved that the cases discharged without evidence of such imperfection are often found after the lapse of months or perhaps years, during which period no rheumatic phenomena have been manifested, to present undoubted evidence of mitral regurgitation. The process of change in the inflammatory products of rheumatic endocarditis—the gradual fibrous transformation, whence the thickening of the valves, cords, and columns, and so the retraction of the valve-curtains—is therefore very slow, and its occurrence is not necessarily marked by symptoms.

When we come, however, to consider the cases generally which present themselves of children manifesting mitral regurgitation, we find that there are many who have presented no evidence whatever of a rheumatic antecedent. For instance, in a series of one hundred and eighteen cases of mitral regurgitation in children, the writer has found an absence of any rheumatic history in forty. In eight cases in this series there appeared to be a definite relation with scarlatina as an antecedent, in six with measles, and in three with both scarlatina and measles in sequence. The evidence of post-mortem examination shows that the changes in the valve-structures induced in the cases in relation with these exanthemata differ in no wise from those brought about by the endocarditis which is in relation with rheumatism. In ten of these cases, where there was no evidence of rheumatism, chorea was manifested. A theory of the probable causation of this form of endocarditis in the absence of rheumatism has just been enunciated, and the consideration of the group will be deferred for the present.

In thirteen cases there was no evidence of any antecedent or probable cause of the valvular imperfection. It is important to note that the signs of rheumatism in the child existent during the rise and progress of valvular disease may be extremely slight. In some cases recent eruptions upon the skin constitute the only obvious sign of the rheumatic condition. Of these *erythema,* especially the form with raised edges and circular or irregular outlines—*erythema circinatum* or *erythema marginatum*—should be particularly noted. More rarely *purpura* may be the only objective evidence, and occasionally recent *eczema* is the sign which induces the suspicion of the rheumatic condition. In such cases endocarditis and even pericarditis may be observed to arise, to continue, and to leave permanent valvular imperfection without any notable symptoms whatever. (Fig. 2.)

FIG. 2.



Annie W., aged ten years. Pericarditis and endocarditis occurring with no urgent symptom whatever, diaphoresis being the only notable sign. Erythema marginatum upon the arms. The outline indicates the area of dulness on percussion. After nine days to-and-fro friction became manifest at *a*. During the following seven days friction disappeared, and systolic apical and systolic and diastolic basic murmurs became manifest. A second attack of pericarditis occurred five months afterwards, attended with no constitutional signs whatever or local pain. Loud pericardial friction. Dulness receded to inner area. Recovery with persistence only of systolic murmur at apex.

In a section of cases a *disorder of the nervous system* is the only declaratory sign in a child who on examination of the heart shows mitral regurgitation. *Chorea* is the most common disorder in these cases, but in some hemiplegia, epilepsy, or signs of cerebral embolism have been noted.

In another group of cases, when there has been no evidence whatever of rheumatism, *disorder of respiration* or of *circulation* has been the only indication of disease. Cough is the most common of such symptoms in the child: this is usually due to intercurrent catarrh, to broncho-pneumonia, or to pleuritis in conjunction with these. In some, extreme wasting coincident with the respiratory difficulties may induce the fear of coincident tuberculosis; but the rule holds good that tubercle is rare when cardiac valvular disease is in existence. Therefore, when a murmur of mitral regurgitation is manifested in a wasting child who suffers much from cough, it is to be remembered that the diagnosis of tubercle should not be given without much consideration.

In another subdivision of the cases of mitral regurgitation the only notable sign has been *disorder of nutrition.* The child is said to have manifested no sign of rheumatism, but is emaciated and in many cases is very anæmic. In such instances, even though it may be asserted by the parents that there has been no obvious dyspnœa, the physical signs may show the existence of pericarditis and the development of endocarditis with a persistent murmur of mitral regurgitation. Inasmuch as the post-mortem appearances in such cases are identical with those which are observed in the subjects of acute and subacute rheumatism, we must conclude that endocarditis, with sometimes pericarditis, may be the *only* expression of the rheumatic condition in the child, that these affections can arise and progress without being evidenced by notable symptoms, and that so the permanent imperfection of the valve may have its origin in a previous rheumatic endocarditis which has been untraced and unnoticed.

In a considerable number of such cases of rheumatic endocarditis in children, when the mitral valve has been rendered incompetent—whether there has been decided evidence of rheumatism or not—the lesion becomes compensated. A sufficient hypertrophy of the ventricles takes place to overcome the difficulties, and growth of the heart follows its normal course. Such are the cases frequently met with in adult life when a systolic murmur is discovered at the apex and yet no discomfort is or has been experienced, the origin of the defect being in some cases traceable to an attack of rheumatism, in others being quite undiscoverable.

In other cases in the child there are no such good results. The cardiac chambers dilate, and, as the growth of the heart and that of the thorax occur coincidently, the præcordial region is bulged forward, the rhythmic action is disturbed and generally the cardiac pulsation accelerated, the child is sensible of præcordial distress, wasting and anæmia are prominent signs, and there are frequently-recurring attacks of broncho-pneumonia.

Complicating the power of the ventricles to maintain compensation is the proclivity of the child to recurring attacks of pericarditis. There was clinical evidence of pericarditis in eighteen cases in hospital out of ninety-nine of mitral regurgitation considered due to a rheumatic form of pericarditis. One in five and one-half, therefore, of all cases manifested pericarditis. But the evidence of post-mortem cases showed that pericarditis was manifest in three-fourths of the cases. In many the pericardium is greatly thickened, often universally adherent to the heart, and in such the muscle is often enormously hypertrophied, and the cavities dilated. The symptoms observed in childhood during the progress of non-compensated mitral inadequacy are very varied. The age of the child has some influence in regard to these. As a general rule, the signs in infants and very young children are chiefly those referable to inanition,—emaciation, anæmia, deformity of the thorax. There are in many cases frequently-recurring attacks of bronchitis or broncho-pneumonia, cough being a prominent symptom. In children after the age of four years symptoms more directly indicating disorder of circulation become manifest. Bleeding at the nose may be cited as one of these. Difficulty of breathing becomes a feature, and in some cases most distressing orthopnœa. Præcordial pain and distress are severe symptoms in some cases, and these may be associated with lumbar pain. Palpitation may be a distressing symptom. Dropsy is by no means uncommon, but it rarely follows the gradually ascending course usual in the adult. The œdema is either more general, or more variable in the site of its manifestation. In cases with œdema or ascites albuminuria is a frequent complication; this may be transient and due to venous congestion, but in the majority of cases it is dependent on the coexistence of inflammation of the kidneys and is a sign of dangerous import. In the later stages of the disease vomiting and diarrhœa may be observed as most serious indications; hæmatemesis occurs in some cases.

II. *Chronic Mitral Inadequacy the Result of Non-rheumatic Endocar-*

ditis.—In a minority of the cases presenting a persistent systolic murmur at the apex the symptoms and course, as well as, probably, the pathological causes, differ from those just described. For example, a child hitherto healthy, and presenting no history whatever of rheumatism or of rheumatic proclivity, is the subject of a sudden terror. An attack by a dog, a severe whipping, a fire occurring in the house, may be cited as instances from actual cases. The desultory movements of chorea are soon after observed to commence, and when the child is brought for treatment a systolic murmur at the apex is discovered. This is usually soft or musical, not harsh, coarse, and loud. After recovery from the chorea the murmur may persist, but the child regains its usual health and none of the signs of non-compensation occur. Of course such constitute only a section of the cases of chorea presenting evidence of mitral regurgitation; in many there is distinct evidence of the previous existence of rheumatism in association with endocarditis; in others, though such evidence is wanting, the signs indicate the rise and progress of an endocarditis which is essentially rheumatic, but in a minority the form of endocarditis observed is quite different from the rheumatic. In the few fatal cases the valves are observed to manifest no general thickening, yet near the lines of contact of the curtains are little bead-like elevations. The heart-muscle presents no deviation from the normal; there are none of the evidences of compensation as seen in the rheumatic variety. In some cases after long persistence the murmur observed has become inaudible. In this form of valvular change the writer is of opinion that the first cause is violence done to the endocardium of the edges of the curtains by the tumultuous action of the ventricle at the time of the fright. The resulting endocarditis is characterized either by small papillomatous outgrowths from the endocardium, or by local abrasions thereof in which little caps of fibrin are deposited.

Prognosis.—If it were only a question of restoration of compensation after a lesion inducing mitral regurgitation in the child, the prognosis would be seldom unfavorable. In a few cases, when the powers of nutrition are very low, the ventricles dilate or the muscle degenerates and the heart fails, but as a general rule, in childhood, compensation is readily established, and with due nutrition and care a fair standard of health is maintained. The difficulties in the way of a good prognosis in infancy and childhood are (1) the occurrence of a slowly progressive change in the valves, and (2) the liability of the child to repeated attacks of endocarditis or pericarditis, or of combined endocarditis and pericarditis.

Treatment.—1. *Rest.*—A question of the first importance in the treatment of mitral inadequacy in childhood is that of the means for inducing quietude and regularity of the heart's action. In many cases rest in the fullest degree possible is essential to the proper treatment; but there are cases in which compensation is fairly established when the policy of rest can be unduly enforced, to the weakening of the cardiac muscle from comparative disuse. In any case presenting evidence of progress of endocarditis or any acute manifestation, rest in bed is absolutely to be enjoined. Even this law, however, is not without exception, for the writer has known cases of pericarditis and endocarditis in their acute stages to occur in children with so little discomfort to the subjects that it has been found necessary to allow the children to be dressed and to walk about the ward. To have insisted on their being restrained in bed would have been to provoke paroxysms of grief or anger of greater danger than the course adopted. In general there is no difficulty in keeping the child in a position of physical rest, and this should be done until the physician is assured that the lesion is compensated, and then gradual exercise (never sudden) should be enjoined. It is by no means necessary for a child with well-compensated mitral regurgitation to be debarred from all athletic amusements. It is advisable that the muscles should be trained gradually by well-regulated exercises: prohibitions from healthful games often do more harm than good.

2. *Warmth.*—This, also, is of high importance in treatment. In cases of mitral regurgitation when there are any signs of progressing lesion, with præcordial pain or discomfort, the application of warm linseed poultices to the heart-region is attended with great relief. When respiratory difficulties are manifest, the poultices should be applied to the back as well as front of the chest: the jacket-poultice is a most useful therapeutic measure. In some cases the poultice may be sprinkled with mustard, or with tincture of belladonna or of opium, or both. The digitalis poultice applied occasionally is often a measure of great benefit:

Take of Digitalis leaves, dried, 2 ounces;
Linseed-meal, 2 ounces;
Water, 1 pint.

Boil the leaves with the water for ten minutes, then add the linseed-meal gradually, stirring constantly; spread the mass on tow, and smear a little olive oil on the surface of the poultice.

In the stage of returning compensation, massage of the chest, gently performed, is very valuable. Great care should be taken that the child, when able to run about, is warmly clad with a woollen material next the skin. This should be uncolored, never dyed with the aniline colors so much in use.

3. *Means to Promote General Nutrition.*—These are important at all stages. When there are signs of acute distress, when appetite is *nil* and vomiting perhaps occurs, supplementary alimentation should be practised. Peptonized enemata are very useful, but the writer, as a rule, prefers a nutritive enema made very simply by shaking together in a bottle two ounces of warm milk with one ounce of cod-liver oil, or an egg with an ounce of hot milk and an ounce of cod-liver oil. Such nutritive enema may be administered three or four times a day. The manner of administration is very important, for this should be very gentle. The mode recommended is to procure a very soft rubber male catheter of the largest size and to adapt to its distal end a small glass funnel; to place the child in

lithotomy position, with knees drawn up and buttocks raised, and then to introduce the end of the catheter, duly oiled, into the rectum. Holding the funnel at a low level, pour in the enema as prepared (two to three ounces), and gradually elevate the former so that the fluid enters the rectum by mere fluid-pressure; then squeeze out the contents of the catheter, by pressure with the fingers from above downward, and gently withdraw. Such mode of alimentation, practised for a few days, often tides over a crisis until food can be taken by the stomach. In some cases it must be continued for long periods.

It is impossible here to discuss the dietetics of the child suffering from mitral incompetence: in stages of compensation the diet need not differ from that which is suitable for a healthy child; in non-compensation, and when intracardiac lesions are progressive, it should be the simple semi-fluid diet of the invalid child, especial caution being taken that the nervous mechanism of the heart be not disturbed by an over-distended stomach.

As regards medicinal means for promoting nutrition in valvular incompetence, cod-liver oil has a very high place; it improves the conditions of anæmia, whilst seldom interfering adversely with the processes of digestion. It is best given finely divided as an emulsion, and in doses of from twenty minims (of the oil) to one drachm three times a day:

Take of Cod-liver oil, 30 minims;
Pure glycerin, 10 minims;
Solution of lime,
or
Mucilage of acacia, to 1 fluidrachm.

Iron, in the form of tincture of the perchloride (♏i–♏v), or syrup of the phosphate or hypophosphite (♏x–ʒi), is very useful, and can be combined with the cod-liver oil in many cases with advantage. In certain cases arsenic (Fowler's solution, ♏ss–♏v) is better than iron, notably in those attended with much nervous perturbation. Sometimes small doses of tincture of nux vomica or strychnine may be added with advantage.

4. *Medicines for the Treatment of the Rheumatic State.*—In many cases of chronic valvular disease the administration of alkalies, especially the bicarbonates of sodium and potassium, seems to be attended with much advantage. Such treatment appears reasonable when, as often is the case, the urine is loaded with urates and contains excess of uric acid. The value of the salicylates and salicin may be open to more question in the chronic conditions of valvular disease, but nevertheless the writer thinks that these drugs are often of great value. It is not unfrequently a matter of difficulty, or of impossibility, to be assured in a case of valvular disease whether slow rheumatic changes are occurring or not; but sometimes a case which does not respond to treatment directed to restore compensation distinctly improves under the administration of salicin or the salicylates. It is thought by some that the favorable influence of these agents is only measured by their power of allaying the *painful* manifestations of rheuma-

tism. This is by no means the writer's opinion, and he counsels, in a case of non-compensated valvular disease when from the non-response to the usual measures it may be conjectured that rheumatic changes are in progress, that the sodium salicylate or salicin in an alkaline mixture be given in doses of from three to ten grains with liquid extract of liquorice. In some cases where a septic toxæmia may be suspected, sodium sulphocarbolate in doses of from five to ten grains should be administered every three or four hours. In some of the cases which begin to improve on the alkaline bicarbonates, the addition of small doses of the iodide of sodium or iodide of potassium is often a distinct advantage. Iron may be combined with alkaline treatment in the form of mist. ferri comp., the saccharated carbonate of iron, or tartrate of iron.

5. *Cardiac Tonics.*—In the treatment of non-compensated mitral regurgitation, *digitalis* stands in the first place for its importance: there appears to be a danger, however, that it may be used too indiscriminately. It is even possible that a practitioner, recognizing in a given case a systolic murmur at the apex, may at once rush to the conclusion that digitalis in some form must be administered. Such a course is much to be deprecated. It may be that compensation is fully established: if so, any agent that alters the rhythm of the heart is to be avoided. It may be that the general methods for promoting nutrition which have been just now sketched may suffice to restore a failing compensation: in such case, also, digitalis is not required. When, however, such means fail, or when there is a distinct call for an agent which shall promote a more perfect systole, then digitalis is of the first therapeutic importance. Especially is it attended with good results in cases where dyspnœa is a marked feature. The drug is usually given in the form of the tincture, in doses of from one to five minims, or the infusion, in doses of from ten minims to one drachm; or the leaves, in powder, in doses of from one-fourth to one-half grain, may be substituted; sometimes one preparation is more efficient than another. Exceptionally digitalin, in doses of from one-hundredth to one-fiftieth of a grain, may be administered hypodermically, and, when digitalis as ordinarily administered may be inert, there may be observed a decided slowing of the pulse almost immediately induced. In some cases, however, digitalis is not well tolerated; in children this intolerance is usually shown by the occurrence of *vomiting*, and it is a good rule always to omit the drug when vomiting is one of the symptoms.

In such cases caffeine or convallaria may be substituted for digitalis. Caffeine may be given in the form of the citrate, in doses of from one to three grains, dissolved in water or in the ordinary saline mixture, or pure, in which case it is best that it be combined with benzoate of sodium, the latter rendering it freely soluble. The best preparation of convallaria majalis is the liquid extract, and the dose should be from four to fifteen minims. In either case it is better that the administration should be interrupted for a day or two after continuous administration for a week; for all these cardiac tonics, though preliminarily increasing the renal secretion, after prolonged action may diminish it. When once, however, it is found that digitalis is well borne, it may be continued, especially in conjunction with iron, for long periods.

6. *Treatment of Dropsy.*—In some cases of mitral regurgitation œdema is transient. It is not unusual for parents to say of the child that "he has the dropsy every week or two," and the expression is based upon truth. An œdema is observed about the face, the ankles, and sometimes the wrists, which disappears after a time. In such cases the treatment by the means hitherto mentioned may so far restore compensation that the symptom does not recur. In other cases dropsy may be a much more formidable sign, and, although in itself only an expression of the morbid conditions of the circulation, may call for special measures of treatment. There may be general anasarca, ascites may be pronounced, and effusion may with rapidity take place into the pleural cavities. In a considerable proportion of these cases renal disease coexists with the cardiac imperfection. The physician should be careful in all cases to examine the urine. This is often a matter of difficulty, for those who have the care of the child often fail to preserve any sample of it for inspection. In such case the physician should insist on examining any linen that may be stained with the urine, and should take the stained portions for examination for blood by the microscope, the spectroscope, or the guaiacum test.

The special treatment of cardiac dropsy in the child may be (1) medicinal, (2) operative. It should be an axiom that medicinal should precede operative treatment, for the disappearance of the dropsy may be rapidly brought about, and this usually occurs through diuresis. The sweat-glands should be made active by measures directed to the skin. Hot-air- or vapor-baths may be administered. In cases where these are not readily available, the surface of the skin should be well sponged over with hot water made alkaline with sodium carbonate (common washing soda), and then the child wrapped in a hot blanket. An alkaline bath daily has been in some cases of very great advantage. The administration of jaborandi or pilocarpine is not to be recommended.

In addition to such diaphoretic means, purgatives are essential. Among such the compound jalap powder, in doses of from five to fifteen grains, holds the first place; or the resin of jalap, in doses of from one-half grain to a grain and a half once in the twenty-four hours, may be substituted. Elaterium in doses of one-twelfth of a grain has acted promptly and favorably in some cases. In addition to these means, a diuretic mixture thus composed may be administered every four hours:

℞ Tinct. digital., ♏ii–♏v;
Spiritus ætheris nitrosi, ♏v–♏xx;
Tinct. scillæ, ♏iii–♏x;
Potass. acetat., gr. iii–gr. x;
Decoct. scoparii, ℥i–℥iv.

The removal of all traces of dropsy in the child is sometimes singularly rapid. When the means fail, it is usually because the renal secretion fails to be stimulated; then the question of operative interference—*i.e.*, of puncture of the œdematous parts or tapping the serous cavities—has to be considered. As a rule, the plan of puncturing the lower extremities, so valuable in the adult, should be avoided in the child. There is in the latter so much tendency to move and chafe the lower limbs that irritation of the points of puncture is readily brought about, with the result of increasing the fretfulness and discomfort. If there be a question between paracentesis and aspiration of the abdomen for coexisting ascites and puncturing the limbs for œdema, the former operation should be preferred. A case may be cited of a boy of seven, who, the ordinary means having failed, and the urine continuing to be very scanty, after the removal of sixty ounces of ascitic fluid by paracentesis voided forty-four ounces of urine in the succeeding twenty-four hours, and rapidly became convalescent.

7. *Sedatives.*—The value of sedatives in the treatment of many cases of non-compensated mitral regurgitation in children is incontestable. It should be a fundamental axiom to use first the least dangerous weapon: so, in the case of a child suffering from palpitation, pain, and præcordial distress, the bromide of potassium or of sodium, in doses of from two to ten grains every four hours, may be tried. In many cases relief is afforded and tendency to sleep induced, and then the intervals between the doses may be lengthened.

In some cases the distress is too great to be thus influenced, and there is very insufficient sleep at night. Then chloral hydrate in doses of from two to four grains may be added to the bromide, or a single dose of from five to ten grains may be administered nightly. In some cases this, too, is inefficient, and recourse must be had to opium. In very young children it is best to give this in the form of compound tincture of camphor. From three to ten minims may be given, according to the severity of the suffering, every three or four hours, and after commencing relief the intervals between the doses may be increased; or Dover's powder in doses of from two to four grains may be given. After six years of age the tincture of opium may be used with less risk of the depressing and sometimes suddenly narcotizing effects which opium in very young children occasionally produces, and in the subject of severe præcordial pain may be continued for long periods with advantage; the doses may be from two to five minims. When opium in any form is administered, the excreta should be watched, and, if there be constipation or absence of bile in the stools, gray powder or small doses of calomel should be administered at intervals.

MITRAL STENOSIS.

Definition.—A pathological condition of the left auriculo-ventricular orifice causing an obstruction to the normal passage of blood from auricle to ventricle.

Pathological Causes.—When in the earliest or slightest degree, the obstruction may be constituted by a ring or fringe of vegetations around the mitral orifice on the auricular aspect: in such cases the cavities and muscle of the heart are not necessarily affected. When in a more marked degree, the curtains of the mitral valve are in part united, their substance thickened, and usually such thickening involves also the cords and fleshy columns: thus in many cases the valve is converted into a membranous funnel, which presents its circular opening when viewed from the auricular aspect. This—the "funnel mitral"—is the usual form. Very rarely in children the aperture is seen to be like a straight slit or a crescent,—the so-called "button-hole mitral." In both conditions the welding and thickening may be extreme, so that the tissue may present the appearance and even the characters of cartilage. In the case of the circular opening this may be so reduced as scarcely to admit a goose-quill, and in the button-hole form the slit may be traversed with difficulty by the smallest silver coin. The left auricle is usually dilated and hypertrophied. Its wall may be nearly as thick as that of the normal right ventricle. In many cases dilatation preponderates over hypertrophy. The left ventricle is generally not larger than the normal, it may be smaller: the exceptions to this rule are when regurgitation preponderates over stenosis. In all cases the right chambers are engorged and dilated. It is noteworthy that in some instances the aorta is smaller than the normal. Pericarditis has been found to coexist in more than one-third of the fatal cases.

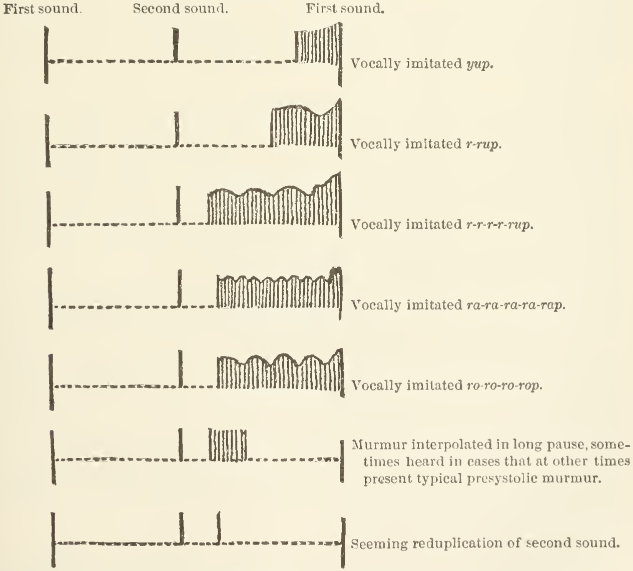
Diagnosis.—The outline of the heart as obtained by percussion may show a disproportionate enlargement of the right chambers, the dulness over the left being not abnormal. This sign, however, is by no means constant, for a past or present pericarditis may cause a general enlargement of the heart's area.

It is important to note if the apex is felt in the normal position whilst the right ventricle is found to beat forcibly below and to the right of the ensiform cartilage. In mitral stenosis the apex is not displaced, whilst in regurgitation it is to the left of the normal.

A sign of high importance is *thrill.* This is to be felt by the fingers or hand lightly applied over the apex: the exact situation is usually somewhat to the right of the apex-beat. When timed by other fingers placed over the apex-beat or the carotid pulse, the vibratory tremor is observed to cease suddenly with the apex-beat or pulse. It is presystolic. Such thrill was noted in seventeen out of thirty-five cases.

The most constant sign of all is the peculiar murmur, whose characteristic is that it abruptly ceases at the moment that the apex strikes the wall of the chest or the pulse is felt in the carotid. This murmur in the child is generally harsh; in some it is rattling, in others rolling, in character; in some cases it commences almost immediately after the second sound, occupying the whole of the long pause, and ending with a maximum of loudness when the apex strikes the chest. In these cases, of course, the murmur occupies both the diastolic and presystolic periods. In other instances the murmur is much shorter and is heard just before the first sound. In such case it is strictly presystolic or auriculo-systolic. In a large majority a systolic murmur of mitral regurgitation is heard in the same subjects, but in eight out of thirty-five of the writer's cases the murmur indicating stenosis alone was heard. The best practical mode of differentiating these murmurs is to ask the simple question, Does this bruit stop suddenly or not? A systolic murmur never stops suddenly, it fades off gradually; a presystolic almost invariably stops suddenly and, as it were, forcibly: scarcely ever do the two murmurs so run into each other as to be indistinguishable. It may so seem to the superficial observer, but on auscultating different points in the neighborhood of the apex a spot will be found where the presystolic comes to its sudden termination, whilst at other spots—usually below and to the left of these—the systolic alone is heard.

DIAGRAM OF AUSCULTATORY SIGNS OF MITRAL STENOSIS IN THE CHILD.


First sound.
Second sound.
First sound.
Vocally imitated *yup.*
Vocally imitated *r-rup.*
Vocally imitated *r-r-r-r-rup.*
Vocally imitated *ra-ra-ra-ra-rap.*
Vocally imitated *ro-ro-ro-rop.*
Murmur interpolated in long pause, sometimes heard in cases that at other times present typical presystolic murmur.
Seeming reduplication of second sound.

Another sign of great importance is the suddenness of the first sound, which resembles the tap made by a hammer. When in a case presenting a systolic murmur such sudden tap is observed, mitral stenosis may be suspected. Another auscultatory sign of importance as aiding the diagnosis of mitral stenosis is a sound resembling a reduplication of the second sound. This is, however, rarely heard at the base, where the second sounds are most audible, but to the left of the sternum and near the apex. The cause of the sound is probably a sudden tension of the mitral following the normal second sound and preceding the presystolic murmur.

There may be thrill without murmur, and murmur without thrill, and both these signs may coincide. It is by a combination of the signs which have been mentioned that the condition of mitral stenosis can be accurately diagnosed. It must be distinctly recognized that in some cases the presystolic murmur of mitral stenosis is a variable murmur,—it may be here to-day and gone to-morrow: when there is either thrill or murmur, or when both occur decidedly presystolic in rhythm, the diagnosis of mitral stenosis is assured.

In a minority of cases the auricular impulse may be demonstrated on the wall of the chest. A pulsation is seen in the second and perhaps third left interspace which precedes the impulse at the apex. These movements are rendered more visible by affixing little levers of thin paper or cotton wool over the points of pulsation. They clearly demonstrate the presystolic pulsation of an hypertrophied left auricle. Good observers have denied the existence of this sign, and it must be allowed that it exists in only a small proportion of cases; but in four out of thirty-five it was very marked, and was demonstrated to most competent and critical witnesses.

Etiology.—It is thought by some that mitral stenosis is in occasional instances a congenital affection. The smooth and even appearance of the septum which exists between auricle and ventricle has probably given rise to the idea that the condition may be the result of an error of conformation. There are, however, many arguments which may be adduced against this view. In the first place, mitral stenosis is very rarely met with in conjunction with developmental anomaly. It is not observed in cases of congenital cyanosis. It is improbable that it should be the result of endocarditis in the fœtus, for in intra-uterine life such endocarditis affects chiefly the right chambers of the heart, and yet in the fatal cases of mitral stenosis any considerable thickening—and certainly disproportionate thickening—about the right auriculo-ventricular orifice is very unusual. Moreover, the condition is not observed in the earliest years of life. The youngest patient in thirty-five cases recorded by the writer was five years of age, and twenty-nine of these cases were between nine and thirteen years of age. It is therefore very improbable that mitral stenosis can in any case be considered a congenital affection.

On the other hand, there is a distinct and undoubted relation between it and rheumatism. In this relation mitral stenosis resembles mitral regurgitation. When we come to inquire as to the degree of intensity of the rheumatism which is the antecedent of the two affections, we find that there are striking differences. Thus, in children who had manifested acute or subacute rheumatism, the proportion of cases of mitral regurgitation to those of mitral stenosis was 64 to 6 (10.6 to 1); in those with no rheumatic antecedents, but with a history of previous scarlatina or measles or both, 23 to 4 (5.7 to 1); in those in whom there was evidence only of rheumatoid pains, 8 to 2 (4 to 1); and in those manifesting no history of rheumatism nor obvious morbid antecedent, 32 to 15 (2.1 to 1). In cases wherein there was no history of rheumatism the changes in the endocardium were in the nature of rheumatic endocarditis, and were sometimes accompanied by pericarditis: in fact, there were no points of differentiation from those cases which were undoubtedly in connection with rheumatism. It would seem, therefore, that the correct conclusion is that mitral stenosis is intimately associated with rheumatism, but more frequently with its insidious than with its violent and explosive varieties.

It is probable that the curtains of the mitral valve become united by adhesions consequent upon endocarditis at those portions which are near the wall of the ventricle, the even pressure of the blood extended from the auricle tending to induce the smooth surfaces and the funnel-like form. Concurrently the tendinous cords and columns undergo a process of thickening; and if this thickening be excessive the "button-hole" rather than the "funnel" aperture is produced.

Clinical History and Progress.—It is only in a limited number of cases that the mode of genesis of the condition of mitral stenosis can be traced by direct observation. The available facts, however, warrant the following conclusions:

1. That a systolic murmur at the apex may in course of time be found accompanied with, or replaced by, a presystolic murmur. The evidence seems to show that such development or replacement is slow and gradual. In one case a systolic murmur carefully observed for four weeks was thereafter found to be preceded by a short presystolic murmur. In other cases a presystolic murmur was found prefixed to the systolic after the lapse of two, three, and four months respectively. In a girl of nine a systolic murmur was found to be accompanied by a presystolic after the lapse of four months, and two years afterwards the systolic had disappeared, leaving a presystolic terminated by a sharp, sudden, and loud first sound.

2. That a presystolic murmur may subsequently be found to be followed by a systolic murmur. In a boy of eight in whom a typical presystolic murmur, abruptly terminated by the first sound, had been observed, there occurred an attack of subacute rheumatism and then a systolic murmur became affixed. Subsequently the systolic murmur increased in loudness and was heard over a wide area, whilst the presystolic was audible only at a point just below and internal to the left nipple. Again, in the case of a girl of twelve in whom a long grinding typical presystolic thrill had been noted, it was observed on one occasion, in the course of an attack of subacute rheumatism, that the sounds had quite altered, a loud *systolic* murmur being manifest at the apex. Subsequently both systolic and presystolic murmurs continued to be audible. Several similar instances might be quoted.

It is difficult to obtain good evidence as to the clinical signs which betoken the advent of mitral stenosis in those cases which occur insidiously without rheumatic accompaniment, but there is a very strong probability of the advent of mitral stenosis if in any case a seeming reduplication of the second sound or—as occurs very rarely—of the first sound is observed.

As regards the symptoms noted in cases of mitral stenosis, it may be said that in about half the number (twenty in thirty-eight) they closely resemble those so frequently observed in mitral regurgitation,—viz., difficulties of respiration, cough, and, in advanced stages, dropsy and the typical cardiac distress.

In the remaining moiety the most noteworthy symptoms are those which indicate lesions of the nervous system, and can in a large number of instances be traced to embolism of a branch of some cerebral artery. For instance, a boy of seven, pale, emaciated, and with projecting præcordium, suddenly manifested right hemiplegia with aphasia. At the autopsy "button-hole" mitral constriction was demonstrated, and the left anterior and middle cerebral arteries were found to be plugged with particles of fibrin, evidently derived from a coagulum attached to the margin of the mitral orifice. In one of the writer's cases a girl of ten became suddenly paralyzed in the right arm: she recovered completely from this affection, and seven months afterwards died after manifesting cardiac distress with dropsy. Stenosis of the mitral orifice was found, with great hypertrophy of the heart. In another instance a boy aged three and one-half was suddenly seized with epilepsy, unconsciousness lasting twenty minutes. Nine months afterwards chorea was manifested: recovery took place, but after a second period of nine months another attack of chorea was manifested. Now on his coming under observation a well-marked presystolic murmur was observed. In a little girl under observation at the hospital, very emaciated, suffering much from dyspnœa and manifesting marked systolic and presystolic murmurs, sudden unconsciousness occurred, and death soon followed. In a boy of five manifesting well-marked presystolic murmur and thrill, a "fit" had occurred eighteen months previously, with such profound unconsciousness that the child was thought to be dead: nine months afterwards chorea developed. In a girl of five with mitral stenosis, there were repeated and well-marked attacks of epilepsy. In thirty-eight cases of mitral stenosis in children recorded by the writer, there were nine cases of chorea: of these, two were right and two left hemi-chorea. It is noteworthy that, with the exception of chorea, not one of these severe lesions of the nervous system occurred in the subjects of mitral *insufficiency*.

Prognosis.—As in mitral insufficiency, the danger in mitral stenosis in children is not measured by the degree of difficulty in obtaining a restoration of compensation. The chief peril to life is that of pericardial or renewed endocardial complication. The liability to pericarditis in children who are the subjects of mitral insufficiency and of mitral stenosis respectively seems to be about equal,—in the writer's cases 1 in 5.5 in the one, and 1 in 5.4 in the other. It is this proneness to renewed rheumatic inflammation of the pericardium and endocardium that largely contributes to the fatality of the affection in early life. A special cause of danger as well as an element of uncertainty in the prognosis of mitral stenosis lies in the liability to embolism. It is probable that in the period of child life mitral stenosis is more dangerous to life than mitral regurgitation, but in a considerable number of cases compensation becomes satisfactorily restored, and, no further development of disease occurring, the subjects attain maturity without any notable symptoms of discomfort.

Treatment.—In the great majority of cases the treatment should be conducted according to the rules already laid down for the management of mitral insufficiency. It may be a question whether, when it is proved to be necessary to administer a cardiac tonic for protracted periods, *digitalis* should be the agent chosen. The writer has found that in some cases of mitral stenosis the administration of convallaria is attended with better results than the treatment by digitalis. It would appear that in the condition of obstruction between auricle and ventricle there is a persistent cause for disturbance of the cardiac rhythm, and that in certain cases digitalis, though increasing the muscular force of the ventricles, tends still further to disturb the rhythm, whilst convallaria, though efficiently aiding the ventricles, does not tend to superinduce irregularity. Striking instances have been seen of the value of convallaria in the treatment of failing compensation in cases of mitral stenosis, but it is only right to say that these are more marked in the adult than in the child.

When *embolism* threatens, it is probable that much good may be done by the administration of ammonia, as suggested by Dr. B. W. Richardson. The difficulty is the detection of such threatenings. If in any case of mitral stenosis under observation a sudden rise of body-temperature occur, not to be explained by the usual causes of fever, embolism may be suspected. In such case ammonia may be administered ; and the best mode is the liquor ammoniæ, in doses of from one to five minims, with liquid extract of liquorice well diluted (as practicable) with water, and repeated at frequent intervals,—every hour or every two hours. The object of the administration of ammonia is to render the blood more fluid and less disposed to the formation of coagula. And, when the symptoms have indicated the occurrence of embolism of a cerebral artery, such administration may not be futile, for, if there be no solution of the clot, the block may possibly be less dense if the blood be rendered less coagulable.

TRICUSPID INADEQUACY.

Definition.—A pathological condition of the right auriculo-ventricular orifice inducing reflux during ventricular systole into the right auricle.

Pathological Causes.—In the child tricuspid regurgitation may be brought about by endocarditis affecting the tricuspid valve, or by a dilatation of the right ventricle preventing due apposition of the valve-segments : such dilatation is induced by disease at the mitral orifice. The post-mortem appearances in cases of tricuspid regurgitation are—

(1) Thickening of the valve-structure: this may be seen in the absence of any evidence of inflammation in cases of congenital malformation when there is undue tension in the right chambers, or it may be associated with definite signs of endocardial inflammation.

(2) Vegetations having like characters with those observed in the endocardium of the left chambers: such vegetations were found in six cases out of thirty-two autopsies in all forms of valvular disease in children. It has been contended by Dr. Byrom Bramwell that endocarditis affecting the tricuspid is more common than has been generally supposed, and that all traces of such inflammation may pass away: the writer is disposed to agree with this view. Certainly tricuspid valve-lesion is not uncommon in children: it was observed in one-fourth of the cases which were examined post mortem. Tricuspid endocarditis especially occurs in intra-uterine life, and is relatively more frequent in very young children.

(3) Dilatation of the right ventricle, rendering the auriculo-ventricular orifice abnormally large and incapable of due closure by the tricuspid valve. This is always the result of overstrain of the right ventricle, either from mitral regurgitation, the regurgitant stream from the left ventricle opposing the force of the right in driving blood into the pulmonic circuit, or from mitral stenosis, when a perpetual obstacle exists at the left auriculo-ventricular aperture to the current from the right ventricle through the left auricle to the left ventricle. Under either of these conditions the muscle of the right ventricle is subjected to abnormal strain, and its cavity tends to become dilated.

Diagnosis.—Tricuspid regurgitation may be detected in the fœtal heart. A case has been recorded by Professor Peter, of Paris. In the case of a healthy girl, aged seventeen, arrived at the normal term of pregnancy, auscultation a little to the left of the linea alba and four finger-breadths below the umbilicus demonstrated, instead of the regular *tic-tac* of the fœtal heart, a loud, rough murmur followed by a short sudden sound like the second sound of the heart. This was considered to indicate a valvular affection of the fœtal heart. The infant was still-born, and the autopsy disclosed endocarditis of the tricuspid, with abundant vegetations and thickening, with retraction of the chordæ tendineæ so that the valve was drawn towards the wall of the ventricle and rendered incompetent to close its orifice. The valves on the left side of the heart were healthy, and there was no congenital malformation.

It may be said, in general terms, that a systolic murmur heard over the apex of the heart in a *very young* infant is more likely to be due to tricuspid than to mitral regurgitation, though these diseases may coexist. In children of a later age the diagnosis of tricuspid from mitral regurgitation from the site of the murmur may be very difficult, for the comparative loudness of the mitral may drown the sound of the tricuspid murmur. It is only in children nearing the age of twelve that one can with any precision differentiate a systolic murmur with maximum at the normal apex (mitral) from a systolic murmur over the base of the ensiform cartilage (tricuspid). Tricuspid regurgitation, however, in the child is not always shown by a systolic murmur in the tricuspid area. Other signs must be sought. Of these the chief are a firm and forcible contraction of the right ventricle felt in the epigastrium and an increase of dulness on percussion over the right chambers. This dulness may extend a finger-breadth to the right of the right border of the sternum. A concomitant sign of great importance is turgescence of the veins of the neck: this is especially seen when the child coughs. Distinct venous pulsation in the jugular is not so evident as in the adult, and, though great enlargement of the liver is a notable and important sign, the writer has not seen an instance of well-marked pulsation of the liver in childhood.

Clinical History and Progress.—In a young infant it is safe to conclude that tricuspid insufficiency is due to endocarditis affecting the valve and its attachments. In later childhood it may be a question whether such insufficiency is due to existing endocarditis or to a dilatation of the tricuspid orifice secondary to disease in the left chambers of the heart. It is, of course, well known that, in the child as in the adult, disease at the left auriculo-ventricular aperture brings about increased tension in the right ventricle, and that as a consequence of such heightened tension the right ventricle tends to become hypertrophied, and, if its muscular power be not adequately sustained, to be dilated. Furthermore, the inadequate propulsion through the arteries tends to undue engorgement of the venous channels and of the right auricle. It may be, therefore, that tricuspid inadequacy is not the result of disease in the valve-structures, but of changes in the capacity of the ventricle. The question, however, whether or not tricuspid endocarditis is present in the child when insufficiency is manifest, is very difficult. It is most probable that the older views, which regarded such passive dilatation as the rule rather than the exception, were erroneous, and that when we meet with signs of tricuspid insufficiency in childhood there is usually a wave of endocarditis with the development of vegetations on the tricuspid as well as on the mitral valves.

Treatment.—It can hardly be doubted that the advent of tricuspid regurgitation imposes new difficulties in the treatment of a case of valvular disease: the dyspnœa is generally accentuated; there is often increased præcordial distress; dropsy tends to increase; cough is frequently more marked, and in the paroxysms of coughing the veins of the neck are observed to be turgid.

In tricuspid regurgitation, from whatever cause arising, whether from acute endocarditis or from consecutive dilatation, the first desideratum is *rest:* so in time the wave of endocarditis may disappear. It is a matter of experience that in tricuspid regurgitation the usual cardiac tonics—digitalis, etc.—fail to manifest good results; the reason is clear, for any increased power of the ventricles causes the right ventricle to force more blood back into the venous channels. It is in such cases that abstraction of blood comes to the aid of the heart-tonic as a useful therapeutic measure. Aspiration of blood from the right ventricle, the right auricle, or the external jugular vein can scarcely yet be said to be a practical therapeutic measure, though much can be said theoretically in its favor. Bleeding from the arm has certainly given good results, but, having regard to the susceptibilities of the little patients and of those who attend upon them, the most feasible method is *leeching.* One, two, or three leeches may be applied over the præcordium every day or every other day, and in many cases the improvement is most decided, so that the treatment is not opposed, but rather welcomed. The tension in the venous channels being relieved, the cardiac tonics previously powerless begin to do their good work.

With the exception of the attempt to relieve the tension in the right chambers of the heart, the treatment of the subject of tricuspid regurgitation should be conducted on lines similar to those detailed in regard to a case of mitral regurgitation. Some cases do well on the syrup of the iodide of iron (♏x–♏xxx). In others arsenic in combination with alkalies is of distinct value.

AORTIC VALVULAR DISEASE.

Definition.—A morbid condition of any of the valve-segments of the aorta, occasioning either obstruction to the current during systole or reflux into the ventricle during diastole.

Pathological Causes.—In thirty-two autopsies of cases of valvular disease in children, lesions of the aortic valves were found in thirteen. Vegetations were seen upon the segments in nine instances; in one there was also destruction of tissue,—*i.e.*, ulceration. In four cases the segments were thickened, and in one only of these there was decided incompetency. In childhood, therefore, the thickening and retraction of the cusps so often seen at later ages are comparatively rare, whilst the recent changes of endocarditis are frequent, although not so common as the mitral lesions.

Diagnosis.—This is chiefly made by the discovery of a systolic murmur with maximum about the third right costo-sternal articulation, sometimes carried in the direction of the arteries, by a diastolic murmur in the same situation or at any spot between base and apex, sometimes obviously conducted in the course of the regurgitant current during diastole; or of two murmurs associated having the above characters. In thirty-five cases in which the diagnosis of aortic disease was made, a systolic murmur was found in twelve, a diastolic in twelve, and combined murmurs in eight. Of concurrent signs a strong pulsation or heaving of the left ventricle is to be noticed with the usual signs of hypertrophy; pulsation at the commencement of the aorta has been observed, and pulsation of the arteries of the neck is a noteworthy sign. The suddenly-collapsing pulse at the radial—the Corrigan's pulse—is not met with proportionally so frequently as in the adult, but in some cases it is very marked. It is to be remembered that the murmur indicating aortic disease may be very slightly pronounced. In one case the second sound was noted to be very ill marked at the base, though there was no murmur; the autopsy demonstrated vegetations on the aortic valves: again, where the second sound was only noted to be prolonged, the aortic segments were not only covered with vegetations but had also suffered distinct loss of tissue. The coexistence with signs of mitral valvular disease is almost invariable. The differential diagnosis is made by the observation of the two areas of audibility of the murmurs.

Clinical History and Progress.—In the great majority of cases of aortic valvular disease in children there is a distinct association with rheumatism, acute or subacute. In a series of thirty-five cases there were but nine exceptions to this rule. The exceptions were four cases of chorea, one of scarlatina, one of measles, and three of doubtful histories.

The proportion of mitral cases to aortic was found to be about four to one. In one hundred and sixty-eight cases of valvular disease in children under the age of twelve, taken from the London Hospital records, thirty-five were aortic: in the writer's cases there were in the subjects of acute or subacute rheumatism forty-nine mitral to twelve aortic. It might seem that the intensity of the rheumatic process had something to do with the production of the aortic lesion, from the fact that, though the proportion of mitral to aortic lesions when there were decided articular signs was four to one, in the cases which manifested only pain without swelling of joints the proportion was only nine to one.

On the other hand, it is proved that the aortic lesion can arise and develop with no notable signs or symptoms whatever. In a boy aged eleven and a half, two months after rheumatic fever pericarditis with endocarditis progressed without any obvious impairment of health, the lad going to school all the time; a basic systolic and diastolic murmur became manifest. In another boy, manifesting signs of subacute rheumatism with systolic and diastolic murmurs at base and systolic murmur at apex, with great cardiac hypertrophy, visible pulsation in third right interspace, and marked Corrigan's pulse, there were no evidences of dyspnœa, no signs of distress or discomfort, the boy expressing himself as "all right."

Clinical observation abundantly proves that the murmurs indicating aortic lesions, whether obstructive or regurgitant, in children may in some cases pass away leaving no trace. Very distinct and musical murmurs may thus disappear, and that without any evidence of embolic plugging. It is quite possible that vegetations may thus be gradually removed from the valves, leaving the latter in conditions indistinguishable from the normal; or the segments may remain thickened, as shown by the post-mortem evidence, without impairment of their coaptation. There can be no doubt, however, that in some cases a serious insufficiency of the valve remains. In these there is often a very great hypertrophy of the left ventricle, which during the period of child-life may maintain compensation.

Sudden death, such as occurs not infrequently in case of the like lesion in the adult, is rare or unexampled in children. The great danger in childhood is not the failure to maintain compensation of the valvular lesion, but the recurrence of acute inflammation in the valves and among the muscular fibrillæ (myocarditis).

Prognosis.—Supposing an aortic murmur to have developed in close relation with acute or subacute rheumatism, the prognosis must be grave, because it indicates a widely-spread endocarditis. Supposing that a condition of aortic regurgitation with or without aortic obstruction be diagnosed in a child long free from any rheumatic sign, the lesion being perfectly compensated, the opinion may be given that, if no renewed rheumatic attack occur, fair health may probably be maintained till the time of adult life, when the risks which then attend regurgitant aortic disease must be estimated. Supposing that the systolic murmur of aortic obstruction, or even a slight musical diastolic murmur, is observed in a child having no history of rheumatic proclivity and no sign of hypertrophy of the left ventricle,—in the subject of non-rheumatic chorea, for instance,—a very hopeful prognosis may be given. The murmur may pass away entirely, or if it persist it may not perceptibly affect the health.

Treatment.—In the rheumatic cases the diagnosis of aortic disease must be taken to indicate a widely-spread area of endocardial inflammation, and therefore still greater care must be taken to avoid overstrain of the heart than in other cases of valvular disease. It is to be remembered also that in cases which may seem to be chronic—*i.e.*, presenting no signs of fever, nor even of discomfort—acute inflammatory changes may yet be in process of evolution in the endocardium. It may be inferred, also, that in such cases it is more than probable that the endocardium is not the only structure involved, but that the myocardium (very frequently) and the pericardium (less frequently) are involved in the morbid process.

The cases, therefore, which present evidences of aortic valvular disease must be kept at rest and under observation until the physician considers that any active change in the valve-structure is improbable. During such time the treatment should be such as is suitable for acute endocarditis. (See the article on acute endocarditis.)

In the cases which are considered to be chronic, presenting signs of hypertrophy of the left ventricle, it is seldom necessary to have recourse to digitalis and other agents which modify the rhythm of the heart. General tonics, and especially iron in the form of the tincture of the perchloride (♍ii–♍v), or the phosphate, or the same with strychnine, are to be recommended. Alkalies with the iron preparations in some cases are more suitable: in such case the latter should be in the form of mistura ferri comp. A convenient and agreeable formula is the following:

℞ Potassii citratis, ℈ii;
Tinct. ferri perchloridi, ♏xx;
Glycerini puri, ʒi;
Aquæ chloroformi, ℥i.
M. Ft. mist.
S.—ʒi–ʒiv ter die.

In cases in which mitral disease coexists where failure of compensation threatens, treatment should be conducted on the lines laid down in the cases of mitral regurgitation. Pain at the heart is best treated by frictions with belladonna and aconite liniment over the præcordium or the application of a belladonna plaster. Antispasmodics, such as ether with spt. vini rectificatus and ammonia, may be administered, and when pain is very severe small doses of opium or morphine may be given. It it seldom that digitalis even in these cases is of any advantage: it generally increases the tendency to heaving and discomfort in the præcordial region. The treatment by alkaline iodides is not so successful as in the cases occurring in adult life. Children also are frequently more susceptible to the effects of iodine than adults, especially as regards eruptions upon the skin. The syrup of the iodide of iron may, however, be given in some cases (in doses of ♏v–♏xxx). Finally, it is in cases of aortic disease that it is especially important to bear in mind the necessity of absolute rest,—even more than in affections of the mitral valves. When, however, the physician is convinced that compensation is fully established and that no inflammatory changes are in progress,—that the child has during a period of several weeks manifested no sign of discomfort attributable to the condition of the heart,—judicious exercise, especially in the fresh air, should not only be permitted, but should be enjoined, the dangers of overstrain being carefully guarded against.

MYOCARDITIS AND CARDIAC ANEURISM.

By J. MITCHELL BRUCE, M.D., F.R.C.P.

Definition.—Inflammation of the walls of the heart.

Myocarditis occurs both as an acute and as a chronic disease. It is usually local; very rarely general. The inflammatory process affects all the elements of the cardiac wall, but is chiefly interstitial,—*i.e.*, it mainly involves the intermuscular connective tissue. The so-called "acute parenchymatous myocarditis" of the acute infective and febrile diseases will be referred to at the end of the present article. Reference will also be made here to acute and chronic aneurisms of the heart, which in children usually originate in myocarditis.

Etiology.—*Acute* myocarditis, a very rare disease, is relatively common in children at all ages, including intra-uterine life.[1] Boys suffer far more often than girls, probably because more exposed to injury and rheumatism. I am indebted to Dr. Quain for a number of valuable statistics, hitherto unpublished, on this subject. Of twenty-five cases of abscess of the heart collected by him, no fewer than fourteen occurred in children of fifteen years and under; the youngest was five and a half: thirteen of the fourteen were boys.

Of the determining causes of acute myocarditis the most important are pyæmia and phlebitis. The former is usually consequent on injury of a joint or bone (acute infective osteomyelitis); less frequently it occurs in connection with caries or chronic joint-disease. Acute rheumatism may set up myocarditis,—in most cases by simple extension from the endocardium or pericardium; and it has been found associated with pericarditis in scarlatinal nephritis.[2] Cardiac abscess may sometimes be traced to ulcerative endocarditis, simple or infective, whether directly (through the lymphatics) or by embolism. Acute traumatic myocarditis has followed a kick over the heart in a child.[3]

The etiology of *chronic* myocarditis is more obscure. Sometimes it is of rheumatic origin, when it occurs either as a simple extension of endocarditis

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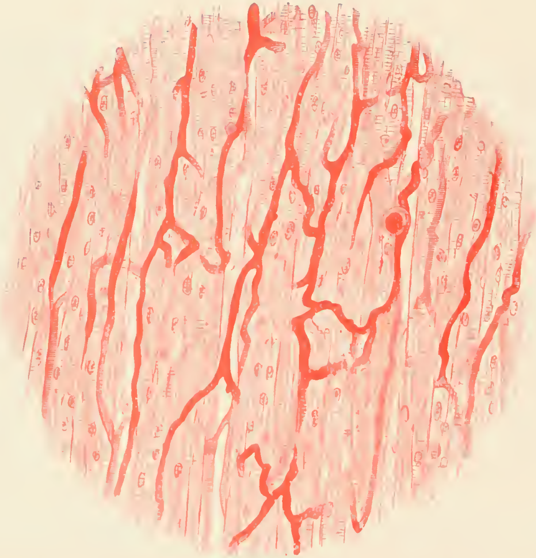
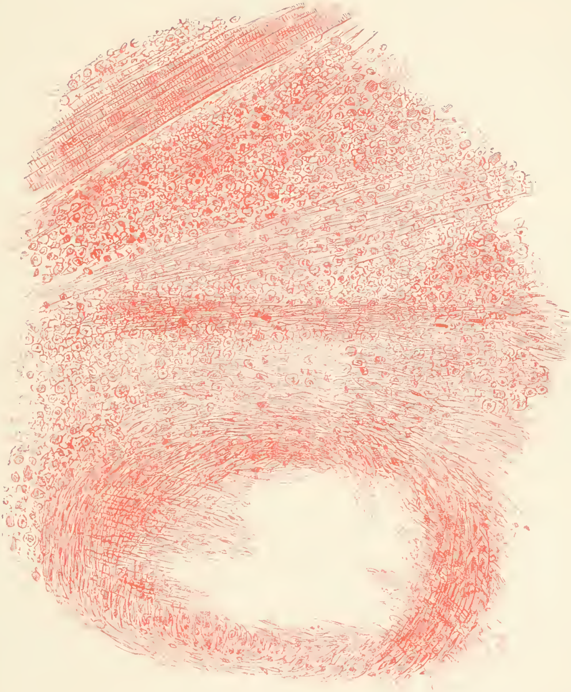
[1] V. Dusch, in Gerhardt's Handbuch der Kinderkrankheiten, 1878, iv. 1, p. 293.

[2] Goodhart, Pathological Transactions, xxxi. 70.

[3] Keating and Edwards's Diseases of the Heart and Circulation in Infancy and Adolescence, 1888, p. 105.



NORMAL MYOCARDIUM, INJECTED. (Drawn by Mr. Collings from a preparation by Dr. Mott.)



ACUTE HEMORRHAGIC MYOCARDITIS, from a case of aortic valvular disease. The connective tissues in the neighborhood of the obliquely-cut arteriole are infiltrated with leucocytes and blood-corpuscles. Many of the muscular fibres have lost their striation, and some are nearly destroyed. (Drawn by Mr. Collings from a preparation by Dr. Mott.)

or pericarditis, or as a fibroid patch and consequent aneurism, referable to local softening from coronary embolism. In other instances it is probably traceable to congenital syphilis. Syphilis also gives rise to unmistakable gummata of the heart, both in the prematurely born and in children up to twelve; and it may well account for a proportion of other less distinctive cases. Chronic inflammation of the heart, as compared with the acute form, is very rare in children. Of fifty-six cases of chronic cardiac aneurism and chronic myocarditis, Dr. Quain found but three in children.

Pathology.—*Acute myocarditis* occurs in two forms,—the diffused and the circumscribed. In the *diffused* form a considerable tract of the wall is infiltrated with inflammatory products and blood-corpuscles, especially the sub-pericardial and sub-endocardial portions, in association with inflammation of the membranes. The *circumscribed* form consists of single or multiple areas of inflammation, usually occupying the left ventricle and the septum.

The acutely inflamed myocardium is either of a dark red, injected, frequently ecchymosed appearance, or of a peculiar mottled yellowish hue; it is softened; and in the localized form it constitutes a swollen focus, which in its later stages becomes grayish red and is finally converted into an abscess

Abscess of the heart bursts outward into the pericardium, inward into a cardiac chamber, or in both directions; septal abscess may establish a communication between the ventricles, or between the left ventricle and the right auricle. Intra-pericardial rupture sets up pericarditis; rupture inward establishes an *acute cardiac aneurism*, which usually has its mouth in the left ventricle, and thus may come to be the source of embolism and pyæmic infection of the other viscera.

Microscopically, acute myocarditis is characterized by infiltration of the intermuscular spaces with an exudation of leucocytes, sero-fibrinous material, and extravasated blood, and by compression and albuminous and fatty degeneration of the muscular fibres. If the process end in abscess, the inflamed area is found to consist of pus, blood-corpuscles, muscular débris, and occasionally infective organisms, which may be traced either to the lymphatics or to emboli in the coronary vessels. (See Plate.)

The liability of acute inflammation of the myocardium to be attended by hemorrhage will be understood by an attentive consideration of the richness and relations of the vascular supply of the walls of the heart, as represented in the plate.

With acute myocarditis there are usually found associated acute endocarditis and pericarditis, and rheumatic or pyæmic lesions in other parts of the body.

Chronic myocarditis is characterized by an interstitial fibrosis,—a growth of the intermuscular connective tissue, and the concomitant degeneration and disappearance of the muscular fibres more or less continuously and completely. The fibrotic process in the heart occupies the usual stages, and presents the familiar appearances, of cellular infiltration, connective-tissue development, and subsequent contraction, as in other organs. (See Plate.) In some instances the inflammation extends inward from an adherent pericardium.[1] More frequently it occurs in spots or areas of various sizes and in different situations, with corresponding effects on the functions of the heart. In its simplest form chronic myocarditis is but a gray, opaque, cicatricial-like condition of the apices of the papillary muscles. In its most pronounced form it ends in the conversion of a considerable area of the ventricular wall into a tendinous mass, which yields to the intracardiac pressure, and becomes first a simple local dilatation and then a true *chronic aneurism* of the heart.

Cardiac aneurism, acute or chronic, is not always secondary to true myocarditis. Embolism of the coronary artery may lead to localized patches of necrosis ("softening," "myomalacia cordis"), with consequent rupture, fatty degeneration, or fibrosis, and either rapid or chronic development of an aneurismal sac. In rarer instances the condition is congenital,—in connection with the pars membranacea of the septum. Chronic cardiac aneurism is met with in the child in a variety of sizes: it may even equal in bulk the whole of the rest of the heart. Its favorite seat is the left ventricle. It appears either as a simple shallow depression in the affected chamber, or as a perfectly developed sac lying in or projecting from the wall of the heart and communicating with the cardiac cavity by a narrow mouth and neck. Pathological records furnish full accounts of the contents of the sac, its relations to the parts around, the state of the endocardium and the pericardium, the occasional termination by intra-pericardial rupture and hemorrhage, the accompanying enlargement of the heart, and the condition of the other viscera, which are frequently the seat of embolism.[2]

Syphilitic disease of the heart takes the forms of fibroid patches in the walls of the ventricles and auricles, gummata, and pericardial adhesion with gummatous involvement of the underlying myocardium. Whilst syphilitic fibrosis is sometimes of inflammatory origin, it is necessary to remember that it may also be the result of specific disease of the coronary arteries, which sets up a softening of the myocardium ("myomalacia cordis") with fatty degeneration and hemorrhage, followed by connective-tissue repair and pigmentation.[3]

Symptoms.—The clinical characters of acute myocarditis are very obscure, being mainly lost in the symptoms and signs of the associated inflammation of the endocardium and pericardium, even in cases of well-defined pyæmic abscess. Thus, in a child of six years, who died under the writer's observation of pyæmic abscess of the heart consequent on acute traumatic periostitis of the tibia, the symptoms connected with the chest

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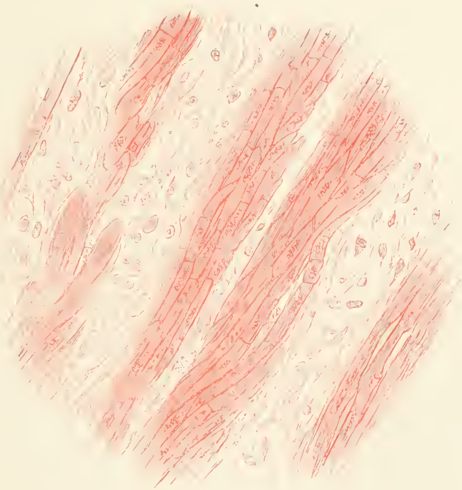
[1] Wilks, Pathological Transactions, xi. 61 (boy of fourteen).

[2] See Pathological Transactions, seriatim.

[3] Mott, Cardio-Vascular Nutrition and its Relation to Sudden Death, The Practitioner, vol. xli. p. 161; Ziegler, Text-Book of Pathological Anatomy, etc., translated and edited by Dr. MacAlister, 1884, vol. ii. p. 46.



CHRONIC MYOCARDITIS.—The various stages of fibroid growth are well seen. The muscular fibres, partly in longitudinal section, partly in oblique, are undergoing atrophy (Drawn by Mr. Collings from a preparation of Dr. Quain's.)

were those of acute pericarditis and left pleurisy, up to a few hours before death, when great distress, twitching of the fingers, sickness, and diarrhœa came on.

An analysis of the recorded cases indicates that there are three leading clinical types of acute myocarditis.[1]

In the first or *cardiac* type the symptoms are those of rapid failure of the heart; anxiety, with pallor or lividity of the face; præcordial and post-sternal pain or sense of pressure or constriction; palpitation; a small, frequent, irregular or imperceptible pulse; faintness or actual fainting; dyspnœa; sickness; frequent sudden action of the bowels, and great diminution in the volume of the urine.

In other cases the prominent symptom has been *dyspnœa*, subjective and objective, with paroxysmal orthopnœa, distressing anxiety and restlessness, coldness and lividity of the extremities, clammy sweats, and periods of unconsciousness. With these the preceding cardiac symptoms may or may not be combined.

The third clinical type of this disease may be called the *cerebral*. With or without the symptoms just described, there comes on a soporose state, preceded by headache, restlessness, occasionally convulsions and delirium, and passing into complete coma. These symptoms, combined with lividity of the face and failure or absence of the pulse, form a very striking clinical picture in the child, reminding us of some cases of fatty degeneration of the heart in the adult, and of the more acute form of ulcerative endocarditis. In several recorded instances in children a pustular eruption appeared on the face, hands, trunk, and scalp.

The *physical signs* of acute myocarditis in the child are ordinarily covered by those of endocarditis and pericarditis. In uncomplicated cases the impulse is very weak and diffused, or imperceptible; the transverse dulness may be occasionally increased; the sounds are feeble, the first being finally lost. Loud murmurs, changing under observation, have been heard in cases of acute cardiac aneurism and perforation of the septum. Local tenderness has also been described.

Chronic myocarditis and chronic cardiac aneurism practically present the same symptoms as chronic valvular disease. In extensive fibrosis of the left ventricle the dilatation sets up the ordinary symptoms of incompetency and backward pressure, with pulmonary and visceral engorgement and dropsy. The histories of cases of chronic cardiac aneurism are characterized by the remarkable absence of other than occasional heart-symptoms, such as fainting, præcordial pain and palpitation, and the occurrence of sudden death from rupture of the sac. This was, for example, the order of events in a case of aneurism of the left ventricle recorded by Dr. Quain in a boy of fourteen.[2]

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[1] Friedreich, in Virchow's Handbuch d. Spec. Path. u. Therapie; Krankheiten d. Herzens, 1867, p. 146.

[2] Pathological Transactions, vol. iii. p. 80.

The *physical signs* are those of cardiac dilatation ; and in some instances of cardiac aneurism the enlargement has been definitely localized. In these cases a variety of murmurs have been described, mainly systolic, as well as a palpable whizzing sensation over the præcordia.

The subjects of congenital *syphilis* of the heart present, in addition to the ordinary evidences of visceral syphilis, the symptoms of cardiac debility, and the signs of cardiac dilatation and possibly of pericardial adhesion without valvular murmur.

Diagnosis.—Acute and chronic myocarditis are among the most obscure of diseases during life : as a matter of fact there are few recorded instances of a diagnosis having been made. This will be, however, more easy in the child than in the adult, in whom similar symptoms may be due to fatty degeneration. The very remarkable association of cardiac abscess with acute traumatic periostitis ought to excite the suspicion of this complication in every case of pyæmia following injury of the bones and joints in children, *however slight ;* and in these, as well as in cases of acute rheumatism and ulcerative endocarditis, a careful study of all the symptoms already described may enable the practitioner to diagnose more or less confidently involvement of the parietes of the heart. These pyæmic cases have been mistaken for acute rheumatism with cardiac complications.

Chronic myocarditis has usually been diagnosed as valvular disease, until sudden death has excited suspicion of the graver lesion.

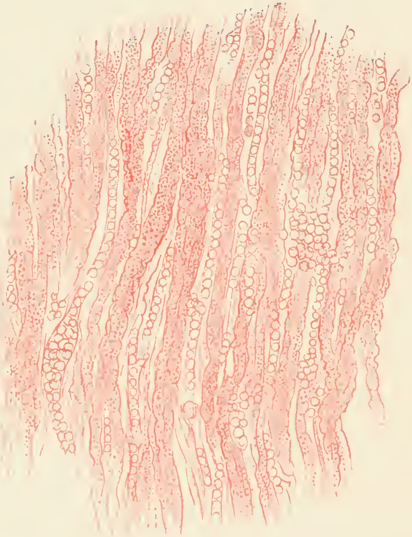
When any peculiarity in the situation and characters of a cardiac murmur in a child suggests congenital disease, the possibility of cardiac aneurism should never be forgotten.

Syphiloma of the heart may be suspected when a child suffering from congenital syphilis with visceral complications presents well-marked symptoms and signs of cardiac or pericardial disease, without evidence of malformation or valvular lesion.

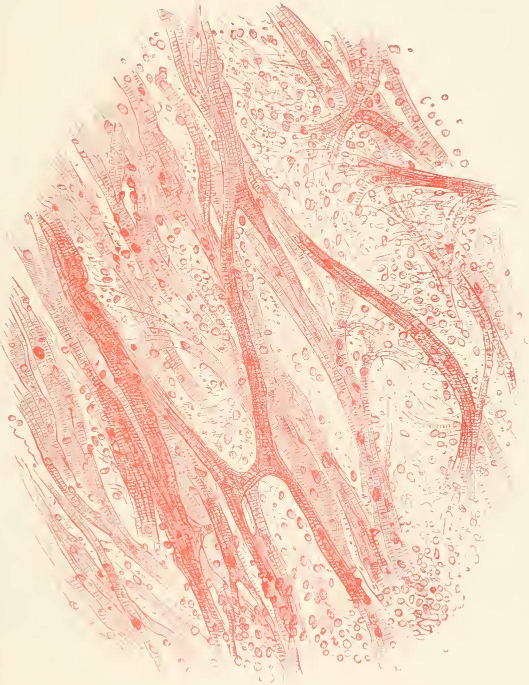
Prognosis, including Course, Complications, and Terminations.—Acute myocarditis, if extensive or ending in abscess, generally runs a rapid course and proves fatal in a few days. When the process is limited to the immediate neighborhood of the lining or covering membrane, no doubt recovery may occur : even abscess is believed to be occasionally cured by absorption, inspissation, and calcification. Death often takes place suddenly without any previous warning of the gravity of the case,—on excitement, or on very slight exertion, such as sitting up in bed. The course of the disease is complicated by the symptoms of intra-pericardial rupture, pericarditis, endocarditis, and secondary embolic infection of the other viscera, as well as by those of the primary lesion in pyæmic cases.

Chronic myocarditis and cardiac aneurism may last for years as a latent or nearly latent condition. The end comes either suddenly, or with the slow development of cardiac failure as described ; in other cases fatal cerebral embolism occurs.

Treatment.—With respect to acute purulent myocarditis, the most



CHRONIC CONGESTION, WITH BROWN PIGMENTATION OF THE FIBRES.—From a case of mitral stenosis with tricuspid regurgitation



SUBACUTE MYOCARDITIS IN CARDIAC FAILURE.—The fibres are atrophied, and the lymph-spaces filled with coagulated lymph and leucocytes; sudden death. (Dr. P. Drakely.)

important consideration is its prevention. The slightest injury of the periosteum in a child must be faithfully attended to, and if fever or articular swelling be discovered the case must be systematically investigated and treated.

When acute inflammation of the heart has actually developed, the treatment will have to be combined with that of pericarditis. Every possible means must be adopted to promote cardiac rest. The child must not be allowed to raise himself in bed, nor to use his arms much. The constant attention of a skilful nurse is therefore indispensable. Food must be of the lightest, least flatulent kind, and given very frequently in small quantities. Very light poultices or fomentations may be used to alleviate the local distress. The sulphocarbolates may be tried in pyæmia. In rheumatic cases the child will be under the influence of salicylate of sodium; and with this, which will have to be given with special caution, must be combined small doses of carbonate of ammonium. The use of digitalis and its allies and of nux vomica, when symptoms of failure supervene, will demand the most serious consideration of the practitioner, the question being whether the softened muscular tissue can bear the strain of stimulation. It is obvious that these drugs, and also ether and alcohol, must be administered in reduced doses at short intervals, so that a sudden and extreme effect on the heart and pulse may be avoided. Paroxysms of cardiac and respiratory distress will call for ether and ammonia, highly diluted. In the cerebral form of acute myocarditis no special advantage is to be expected from remedies directed to the brain, beyond cold applications and constant attention, as the head-symptoms are mainly referable to cardiac failure.

Chronic myocarditis, if it be diagnosed, may be treated on the same principles as valvular disease. The avoidance of exertion is manifestly all-important.

Syphilitic disease of the heart, when it can be recognized, is always associated with grave lesions of the same kind in other viscera for which specific treatment is being employed.

ACUTE PARENCHYMATOUS MYOCARDITIS.

Synonymes.—Acute parenchymatous degeneration, Albuminous degeneration, Febrile softening of the heart, Infectious myocarditis.

Under these names there has been described from time to time a kind of acute change in the muscular tissue of the heart which occurs in acute febrile and infective diseases. The opinions of pathologists of the nature of this disease have long been and still are conflicting, some maintaining that it is truly inflammatory, others that it is degenerative only.

Etiology.—"Parenchymatous myocarditis" is the result of acute febrile and infective processes, such as scarlatina, diphtheria, variola, typhus, typhoid and relapsing fevers, septicæmia and pyæmia, more rarely measles. The condition may be set up during the later as well as the earlier stages of these diseases, or even during convalescence. Phosphorus-poisoning, scurvy, and purpura induce a closely-allied condition of the muscular tissue, in which, however, fatty degeneration is the most prominent change. Severe hemorrhage,—for example, umbilical bleeding in the new-born child,[1]—and impaired nutrition from local causes, such as coronary embolism and acute and chronic pericarditis, also give rise to patches of fatty degeneration, variously associated with myocarditis.

Pathology.—In the acute specific fevers the heart is sometimes distinctly dilated. The myocardium is of a dirty grayish-red or grayish-yellow color, with occasional extravasations; its consistence is soft; its substance is lax, flabby, and friable. Thrombi may be found in the ventricles. Microscopically, the muscular fibres are swollen, their striation more or less lost and replaced by granular (albuminous) and fatty molecules; occasionally they undergo waxy degeneration (Zenker). Along with these evidences of degeneration there are found certain appearances which suggest *regeneration.* The muscle-nuclei are swollen and multiplied, myoplastic bodies occupy the intermuscular spaces,[2] and along with these are a variable number of inflammatory cells (leucocytes) and red corpuscles. Further, the blood-vessels are congested and thrombosed, and the arterioles are the seat of obliterative endarteritis. Corresponding changes are found in the voluntary muscles.

The pathological connection between this acute parenchymatous change and its causes is still unsettled. It may be the result of the specific action of the several poisons on the protoplasm, or of the pyrexia, or of both. It is closely related to fatty degeneration of the heart: indeed, if the destructive part of the process be in excess, it rapidly proceeds to fatty degeneration, which then covers or takes the place of the other changes. Thus it happens, on the one hand, that the acute parenchymatous changes in the myocardium which we have described cannot always be distinguished from the effects of anæmia, scurvy, and purpura on the same tissue, and, on the other hand, that fatty degeneration of the muscular fibres frequently accompanies ordinary interstitial myocarditis, both acute and chronic.

Symptoms, Diagnosis, and Prognosis.—The symptoms directly referable to parenchymatous myocarditis and degeneration are even more obscure than those of the interstitial form, and for the same reason,—namely, that they are lost in the symptoms of the primary disease. Cardiac failure is the chief evidence of this condition of the myocardium. Either slowly or suddenly a child suffering, for instance, from diphtheria

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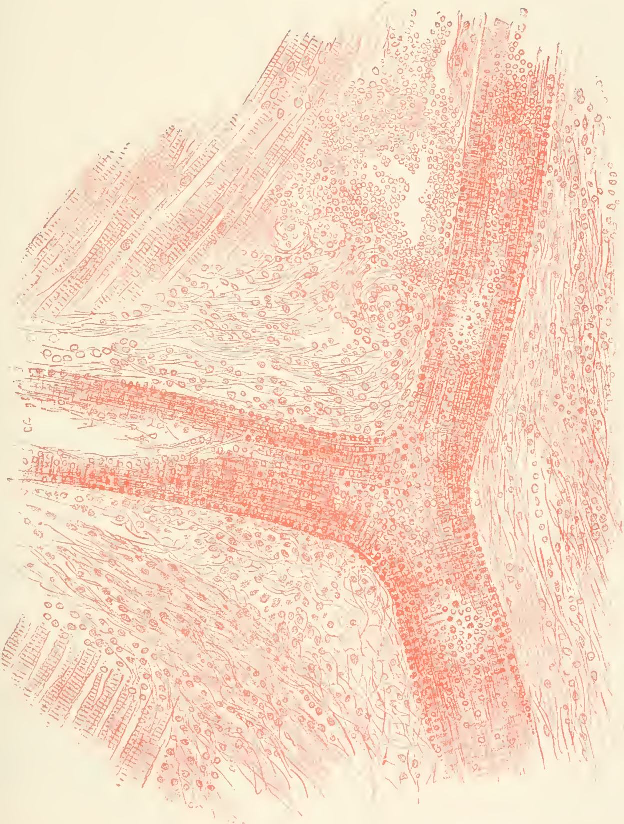
[1] V. Dusch, op. cit., p. 306.

[2] Hayem, Archives de Physiologie norm. et path., 1870, tome iii. p. 274; Huguenin, Revue de Médecine, October, 1888, p. 1002.



Acute Rheumatic Myocarditis, associated with Endo- and Pericarditis.—To the naked eye the myocardium was "fatty" only. The tissues around the artery, seen in longitudinal section, are infiltrated with leucocytes, and hemorrhage has occurred in the upper part. A case of sudden death. (Drawn by Mr. Collings from a preparation by Dr. Mott.)

falls into a condition of collapse. The pulse fails at the wrist, becoming feeble, small, irregular, and either very frequent or remarkably infrequent. The countenance is pallid, with some lividity, and expressive of apathy,—not greatly distressed, with pain and dyspnœa, as in ordinary acute myocarditis. The cardiac impulse and the first sound become weaker and may disappear. Galloping rhythm or a systolic murmur is sometimes developed.[1] The extremities are cold. The skin is bathed in sweat. The urine contains albumen. Death occurs in most cases,—either slowly, with hypostatic visceral congestions, increasing dyspnœa, and asphyxia, or suddenly by cardiac arrest. Recovery is, however, possible.

The *diagnosis* mainly rests on the association of these symptoms with an acute infective disease, especially diphtheria.

Treatment.—The appearance of symptoms and signs of cardiac failure in the course of fevers has long been regarded as an indication for stimulants. These, with proper feeding and the most watchful nursing, are the chief means at our disposal for combating so-called parenchymatous myocarditis. The skilful practitioner, indeed, anticipates these complications, in ordering his treatment, from the first. He is careful not only to secure, as far as possible, abundant nourishment, but also to avoid the abuse of depressant measures, including emetics and pilocarpine. The child must be spared the very slightest exertion and excitement,—an end which, most unfortunately, it is almost impossible to attain in diphtheria if the throat and the feeding are faithfully attended to. Nutrient enemata or suppositories will then be called for. Rest during convalescence from diphtheria may be equally important, for the same reason.[2] When the condition becomes grave, hypodermic injections of brandy, of ether, or of a combination of caffeine and benzoate of sodium, must be given;[3] and, indeed, these and digitalis, strophanthus, or sparteine may be administered without waiting for positive signs of failure of the heart.[4]

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[1] Huchard, quoted by Hayem, loc. cit., p. 576; Leyden, Zeitschr. f. Klin. Med., Bd. iv. p. 346.

[2] Leyden, loc. cit., p. 347.

[3] Lewis Smith, Sudden Heart-Failure in Diphtheria, and discussion at New York Academy of Medicine, Medical Record, November 10, 1888, p. 574. Huchard, Journal de Médecine de Paris, June 10, 1888, p. 920.

[4] Jacobi, Archives of Pediatrics, March, 1889, p. 139

DISEASES OF THE PERICARDIUM.

By T. M. ROTCH, M.D.

In considering the diseases of the pericardium in infancy and childhood, it has been thought wise to submit to the reader, for the purpose of brevity and to avoid repetition, mainly those facts which are distinctive of these diseases, as differing from those which have already been dealt with in adults by previous writers.

The diseases in general will be spoken of only so far as is necessary to elucidate the subject, and mention will be made of certain points which, although pertaining to older subjects as well as to younger, have not heretofore been sufficiently dwelt upon. Free reference to and use of the various articles on this subject have been made, and, as want of space prevents its appearing in the present work, the reader is referred to the excellent bibliography which so thoroughly covers the literature of the pericardium up to the year 1878, compiled by Dr. Franz Riegel in Gerhardt's "Handbuch der Kinderkrankheiten." The writer is also especially indebted to Drs. J. M. Keating and W. A. Edwards, also John B. Roberts, of Philadelphia, for much valuable information contained in their writings.

The anatomy of the infant's pericardium, so far as could be determined by the writer from a dissection of sixteen infants of various ages, appears to approximate, in its relations to the diaphragm, lungs, heart and great vessels, ribs and sternum, so closely that of the adult that there is nothing distinctive to note concerning it. The amount of fluid which normally occurs in an infant's pericardium, although a variable quantity, is probably under five grammes.

PERICARDITIS.

The most frequent disease of the pericardium is pericarditis. It can occur at all ages, but is less common the younger the subject. It has been found in the fœtus and in the new-born, and well-marked adhesions of the pericardial surfaces were observed in an infant dying thirty-six hours after birth.

Etiology.—The etiology of pericarditis in the young is somewhat wider in its scope than in adult life. The prolific source of pericardial inflammation, rheumatism, though not so common as in adults, and assuming a much more subacute type than in young adults, gives rise, in proportion to its frequency in children, especially after the third and fourth years of life, to as much peri-endocardial disease as at a later period. In children, as in adults, these inflammatory lesions may appear before the rheumatic element has declared itself elsewhere, and the intensity of the arthritic pain and the number of joints affected do not correspond to, or rather do not influence, the frequency of the pericardial complication.

In the new-born, pericarditis may be the result of a septicæmic condition following phlebitis, or the absorption of putrid material from the cord, thus resembling the pericarditis which is likely to accompany pyæmia at all ages. It also at times follows periostitis and ostitis in young children, probably here also being associated with septicæmia.

Pericarditis may secondarily be caused by any of the eruptive fevers, but of these diseases scarlet fever appears to play the greatest *rôle* from an etiological stand-point. The occurrence of pericarditis in the above-mentioned fevers may probably be explained by the great tendency of the serous membranes to become affected under such conditions; also by the accompanying congestion of the kidneys, which, together with the prostration following the disease and the readiness with which the surface of the body, under these circumstances, is affected by changes of temperature, renders the pericardium especially susceptible to inflammation. In scarlet fever the pericarditis, when it occurs, usually appears in the second or third week, at a time when the kidneys presumably are not working well and when there is an insufficiently free elimination of the scarlet-fever poison. In addition to these causes, the pericardium shows in childhood a great liability to be influenced by disease elsewhere; and this is exemplified by the frequent complication by pericarditis of tuberculosis of the pleura, especially when it is the left side that is affected, thus showing the additional influence of contiguity.

Inflammation of the pericardium is also quite frequently associated with pneumonia in children.

Pathology.—Pericarditis may be circumscribed or diffuse, and there appears to be no essential difference between the pathological conditions affecting the young subject's pericardium and those which occur at maturity.

The pericarditis sicca of the adult is comparatively unusual in the child, in whom, as a rule, effusion of greater or less extent almost always takes place. The effusion may be sero-fibrinous, hemorrhagic, or purulent.

The tendency to effusion in the child is not only greater than in the adult, but its formation is also characterized by a greater rapidity, and, following the general rule of effusions in young subjects, it is more likely to be purulent than in adults. A slightly bloody tinge to a pericardial effusion is not uncommon in early life, and does not necessarily have the significance clinically which would be derived from a pronounced hemorrhagic effusion.

The white, opaque thickening of the inner pericardial surface, or the so-called milk-spots, so frequently found in adults, are rare in children, but have been found at all ages, and where there is deformity of the chest, as in certain cases of rachitis, they have been especially noticed.

Tuberculosis of the pericardium, as a primary disease, is even more rare in the child than in the adult, in whom it is at times found in connection with caseous bronchial glands. Tuberculosis secondary especially to tubercle of the pleura may occur.

The younger the subject the less likely are there to be adhesions between the pericardium and pleura,—an important fact, to be taken into consideration later, in speaking of the diagnosis of pericardial effusion in the young.

Symptomatology.—Pericarditis may be acute or chronic, primary or secondary.

The subjective symptoms which represent the pericarditis of infancy are very indefinite and unsatisfactory, and even in the child this latency of the early symptoms is so marked and occurs so frequently that it may be said to be characteristic of the symptomatology of pericarditis in early life.

It is so difficult to locate pain when it occurs in the young subject, and a tumultuous action of the heart with general circulatory disturbance is so commonly the result of a diseased condition outside of this central organ, that it is impossible to formulate a practical general symptomatology for the onset of the disease.

When, however, the disease becomes more pronounced, the sensation of dyspnœa and the accompanying orthopnœa, as in the adult, assume a prominent position, and are especially valuable, as they represent a stage of the disease when a decided and intelligent treatment is often of the utmost importance.

Large effusions appear to affect the functional activity of the heart more rapidly in children than in adults, and to occasion earlier the signs of disturbance of the circulation, even although, as in idiopathic cases, there are no complications present.

Diminution in the amount of the urine in cases of pericardial effusion, with a corresponding increase in the urine as the effusion decreases, has been noticed in children.

The physical signs of pericarditis, with few exceptions, are the same as in the adult; but these exceptions are of great importance for diagnosis, and should be carefully considered, for where a friction-sound is absent the determination of a case of pericarditis in a young child presents at times almost insurmountable difficulties.

Owing to the flexible thorax of the child, there is a greater opportunity for the neighboring parts to yield before the pressure of an effusion, and we are thus more likely to have bulging of the intercostal spaces and on inspection a visible alteration of the cardiac area than in adults.

We must also consider that, owing to the small size of the child's thorax, the heart and pericardium are much nearer the anterior surface of the thoracic cavity than is the case with these organs in the adult, and that this occurs both normally and in diseased conditions, especially where there is flattening and thus levelling of the chest. Under these conditions the heart and pericardium are brought in such close contact with the examiner's ear that on palpation he will feel the heart's impulse, and on auscultation hear the heart-sounds, in a more advanced stage of the effusion than would be possible in the adult with a proportionately large increase of the fluid.

It is held, also, by some writers, that in early life the sounds on auscultation in pericarditis and endocarditis at times closely simulate each other. The most important physical sign, when the friction-sound has escaped detection, both for determining whether pericarditis is present and also, when the disease is established, as a guide to prognosis and treatment, is the percussion. A greater diversity of opinion has, however, arisen regarding this sign, and more obscurity has consequently enveloped it, than would seem compatible with the small area of the chest it has to deal with and the progress which it has made in determining the presence of effusions elsewhere. The writer, judging from his anatomical and clinical experience, which has been especially brought to bear on this subject during the past ten years, has come to the conclusion that this diversity of opinion arises from a misapprehension of the anatomical and pathological conditions, which, underlying the clinical phenomena, would, if properly studied and appreciated, elucidate the subject and explain the reason for the diversity of phenomena which undoubtedly arises. It is not that the clinical observers are either incompetent in their ability to observe or incorrect in the reports of their observations, for, on the contrary, they are undoubtedly correct. An entirely insufficient number, however, of properly conducted anatomical investigations have been made to warrant the percussion-rules for a pericardial effusion which have been deduced from the reported cases. The observations from which these rules are derived have in many instances been made on exceptional cases,—exceptional not only in the sense of a diversity of anatomical conditions resulting from pathological causes, but also as differing from observations made on subjects where, from an absence of pathological conditions outside of the pericardium, we can assume that the area of percussion-dulness can be taken as the standard and as representing the typical case with the aid of which the exceptional cases can be studied.

The writer believes that he has already accomplished something definite and exact in the determination of the area of dulness in the typical uncomplicated case of pericardial effusion. He also thinks that the same method which was employed in determining the area of dulness in the typical uncomplicated cases should be adopted in studying the complicated ones. With this end in view, he considers that it is worth while briefly to describe what he has found to be the best method for studying a pericardial effusion on the cadaver, hoping that others, where an opportunity presents itself, may continue this study and publish their results.

In effusions of exactly the same amount the area of dulness may differ, owing to the difference in the elasticity of the lungs and the presence or absence of adhesions. The greater the elasticity of the lungs and the fewer the adhesions, the more regular will be the outline of absolute dulness[1] and the greater its significance as compared with that of the relative dulness,[2] while the reverse of this proposition is true of the relative dulness. Thus the absolute dulness is determined by the retraction of the borders of the lungs, which withdraw from the chest-walls as the effusion gradually distends the pericardium. The enlargement of the area of relative dulness is due to the distended pericardium compressing the lungs, which may be held more or less in position by adhesions. Again, the greater the elasticity and the freer the displacement, the greater will be the compression. Thus the relative dulness with its necessarily irregular outlines, representing extraneous pathological conditions, must especially be investigated in studying the complicated cases, while the absolute dulness should be made use of in determining the typical uncomplicated pericardial-effusion outlines. The older the patient, the more likely is the existence of adhesions and pulmonary morbid processes, which will alter the elasticity of the lung; and the writer, taking these facts into consideration, has concluded that the infant presents the most favorable conditions for determining the percussion-outlines of the typical uncomplicated case, and that the absolute dulness is the most valuable physical sign of effusion in infants and children. Due allowance must be made for the relatively smaller size of the infant's sternum to that of the adult's, this proportional difference being particularly well marked between the child and the male adult.

The number of clinical observations on infants is not yet large enough to provide us with sufficient data from which we can make precise deductions, but the experiments on which the following diagrams of pericardial effusion are based were made on sixteen infants, in none of whom did adhesions exist. In all of these presumably typical cases absolute dulness was found to the right of the sternum, while, to illustrate the difference of percussion-dulness which arises in complicated cases, I would cite the case of an adult at the City Hospital, where, although the pericardium was much distended with fluid, the percussion failed to show dulness to the right of the sternum, and the autopsy revealed adhesions binding the lung tightly to the right edge of the sternum. In this case the effusion was behind the lung, which resulted in resonance being found in an area which with the same amount of effusion in an uncomplicated case would have presented dulness. It is evident, therefore, that we must first study and acquire a precise knowledge of the uncomplicated cases before we are prepared to elucidate those which are complicated by pulmonary adhesions. There is, however, a strong probability that many of the clinical observations made on adults by various competent clinical observers, and tabulated by them as presenting rules for diagnosis, are, from the presence of adhesions, sometimes in one place and sometimes in another, rendered of little practical value, as proved by the difficulty in making a diagnosis by these rules in new cases.

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[1] Absolute dulness means entire absence of resonance.

[2] Relative dulness means diminished resonance.

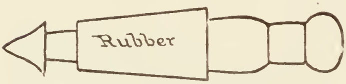
In this connection, also, a case which came under the writer's notice, of an adult with pneumonia of the middle lobe of the lung, is of significance. The solidified lung-tissue came close to the right edge of the sternum, and, by not yielding to the pressure of a pericardial effusion which was also present, prevented the dulness from the effusion, which in an uncomplicated case would have been present in this region, and thus obscured the diagnosis. It is evidently important, therefore, to experiment with artificial effusions on the cadaver, to determine on rules for the diagnosis of a pericardial effusion where morbid conditions of the right side of the body interfere with the usual percussion-outlines found in a typical uncomplicated case. Such conditions are represented by pneumonia, right-sided pleuritic effusions, enlarged liver, enlarged heart, etc.

Various methods of introducing fluids into the pericardium through the sternum have been tried, and failed to give satisfactory results, as, although by dividing the sternum in the median line the pericardium can be entered without perforating the pleural cavity, yet by this method the results of percussion are rendered void, by air not only entering the anterior mediastinum, but also getting into the pericardium itself. The method which was finally devised by the writer, and found to be most satisfactory in its mechanism, was the following.

The subject was placed in the position of orthopnœa,—that is, the trunk was bent upon the lower limbs at an angle of about one hundred and twenty degrees. Tracheotomy was performed, and a clamped rubber tube attached to the glass tracheal tube. The lungs were then inflated through this tube until on careful percussion the absolute area of cardiac dulness corresponded to that delineated in Luschka's plate and verified by Ferber, Sibson, Schroetter, and others, which represents the relation of the parts as it occurs in expiration. This gives an area of absolute dulness which begins at the junction of the upper border of the fourth left costal cartilage, extends downward and outward to the left in rather a curved line, with the convexity outward and keeping two or three centimetres within the nipple (in the average adult), until it joins the dulness of the left lobe of the liver; from the same starting-point at the fourth cartilage it extends down the left parasternal line, or perhaps a little within that line towards the middle of the sternum, until it reaches the liver. It is thus seen that the absolute dulness of the heart is determined, not by the shape of the heart itself, but by the marginal lines of the lungs, varying according to their expansion or retraction; and this is a point which it is well thoroughly to understand at once,—namely, that the pericardium itself, whether it is distended with fluid or not, does not by its own shape, as delineated so often in the plates illustrating pericardial effusions, aid us materially in determining the shape of the area of absolute dulness in a pericardial effusion, but that this area is marked by the retracting or rather displaced borders of the lungs. After the inflation was accomplished, the tracheal tube was clamped so as to retain the lungs in position.

An incision was then made in the median line of the abdomen from the pubes up to within two centimetres of the ensiform cartilage. The liver and stomach were gently drawn away from the diaphragm, and, on palpation of the central tendon of the diaphragm four centimetres to the left of the median line, the heart was felt. This part of the diaphragm was then carefully drawn down away from the heart, and a dagger-pointed trocar pushed through the diaphragm into the pericardial sac, which is adherent to the diaphragm at this point. A full-sized section of the trocar which, after many failures with other instruments, was finally devised and found satisfactory by the writer, is shown in Fig. 1. It is made of brass, with a conical point, and a round shoulder forming the base of the cone, so that, although it easily enters the pericardium, it is difficult to withdraw it, thus acting like a fish-hook. A short piece of rubber tubing, fitting tightly to the neck of the trocar, can, as soon as the point and shoulder have entered the pericardium, be pushed up tightly against the under side of the diaphragm, thus holding the trocar in position, the diaphragm being firmly compressed between the shoulder and the rubber tube and thus preventing the entrance of air.

Fig. 1.

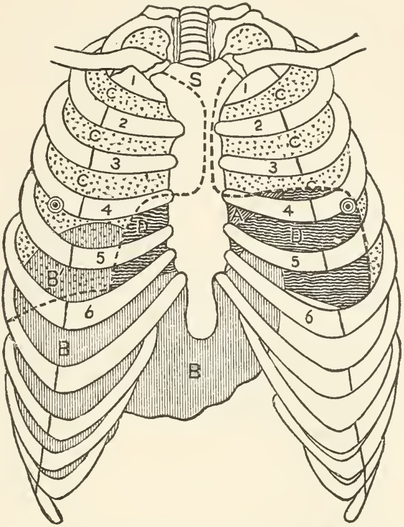


Artificial pericardial effusion trocar. (Rotch.)

The trocar is connected, by means of a piece of rubber tubing (also provided with a clamp), with a simple wash-bottle graded for cubic centimetres and containing melted cacao-butter. Before introducing the trocar the cacao-butter is allowed to fill the tubing and the trocar, so as to displace the air. As soon as the trocar has entered the pericardium the tracheal tube is unclamped, in order that the lungs may be free to retreat before the fluid. When sufficient fluid has entered the pericardium, which is indicated by the graduated bottle, the cacao-butter tube and the tracheal tube are again clamped, the thorax is carefully percussed, and the line of absolute dulness is marked in ink. After twenty-four hours the sternum is removed from above downward, remaining attached below, and we find the lungs in position surrounding the hardened fluid, as represented in Fig. 2, where a rather small amount of fluid has been introduced. By replacing the sternum, and comparing the lines previously marked in ink, by means of needles, with the lines of lung-margins around the effusion, we obtain an accurate result regarding the shape of the area of absolute dulness with this amount of effusion.

As seen in Fig. 2, the area of dulness, in an adult, where from seventy to eighty cubic centimetres of fluid had been introduced, showed a slight increase in the vertical as well as the transverse dulness, and the curved line which bounded the area of dulness was found to start at the sixth rib, four centimetres to the right of the sternum, pass upward to the junction of the fourth cartilage with the sternum, impinging on the lower part of the third left interspace, and then, descending just outside of the mammary line to the sixth rib, pass inward to meet the liver-dulness below, as shown in the diagram. This line marking the dulness was, as is seen in the diagram, an irregular semicircle, with a shorter radius to the right of the sternum and a longer one to the left.

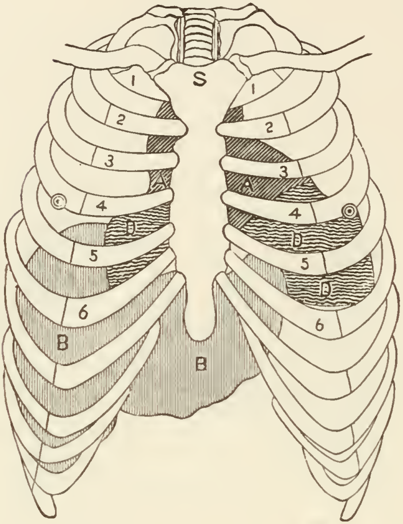
Fig. 2.



Small Amount of Liquid introduced into Sac (Rotch).—▨ A, the portion of the area of absolute dulness which is still caused by the physiological dulness of the heart; ▥ B, liver; ▦ B′, that portion of the liver which is covered by the right lung; ░ C, lung; ▤ D, effusion; A + D, area of percussion dulness found when the effusion is small; S, sternum: ◎, nipple; 1, 2, 3, 4, 5, 6, ribs; - - - - - (broken line), border of lung.

It now becomes of some importance to understand what the above area of absolute dulness was caused by; and this will be best understood by referring to Fig. 3, where in this same subject the lungs have been removed, leaving the heart and pericardium, with its effusion, exposed to view. It will here be seen, on comparing Figs. 2 and 3, that a small section of the dull area, corresponding to the junction of the third and fourth ribs with the left side of the sternum, is formed by the heart itself being free from effusion at this point, while the rest of the dulness is produced by the effusion. On examining also the hardened cacao-butter cast, it was found that the layer of fluid was very thin all over this upper portion of the effusion in the region of the fourth rib and fourth interspace, while the thickest mass of the effusion was, as would be expected from the laws of gravity and the shape of the pericardium, in the lower part of the sac on each side of the sternum in the fifth interspaces, the cast riding the arched diaphragm like a saddle, and the larger part of the mass being on the left side. These points should be carefully noted, as they are significant for diagnosis and treatment.

Fig. 3.

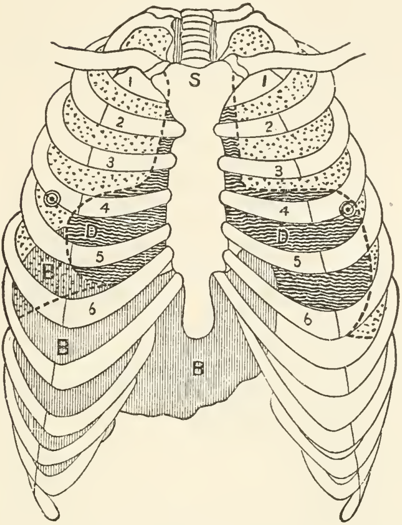


Represents Fig. 2 with the Lungs removed (Rotch).—▨ A, portion of the normal heart enclosed in the pericardium; ▥ B, liver. ▩ D, effusion as it appeared in the sac, the cacao-butter being in small amount, and the lungs having been removed after the butter had hardened; S, sternum; ◎, nipple; 1, 2, 3, 4, 5, 6, ribs.

The same result as to the area of dulness was obtained with a proportionately small amount of fluid in an infant about two weeks old, and out of eighteen injections, mostly of infants of various ages, the percussion-areas of dulness were identical, and in all these cases the lungs were normal and there were no pulmonary or other adhesions.

Fig. 4 represents the position assumed by the margins of the lungs, and the resulting area of absolute dulness, where the pericardium was distended with a large amount of fluid, covering the entire heart: it need not be described, as it speaks for itself. Fig. 5 is drawn directly from the same subject with the lungs removed, and represents also the heart and great vessels in relation to the ribs and sternum, before the pericardium has been distended with fluid.

FIG. 4.

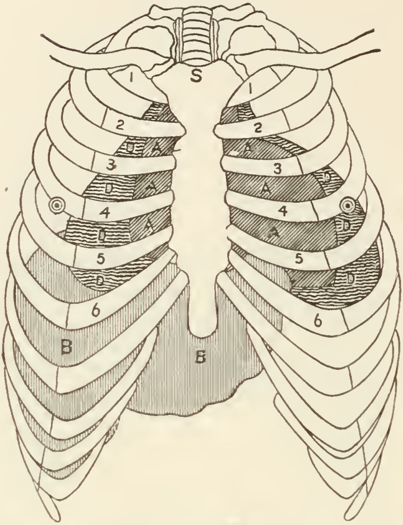


A LARGE AMOUNT OF LIQUID HAS BEEN INTRODUCED INTO THE SAC (Rotch).— ▥ B, liver; ▦ B′, that portion of the liver which is covered by the right lung; ⬚ C, lung, ▤ D, the area of percussion-flatness caused by a large effusion; S, sternum; ◎, nipple; 1, 2, 3, 4, 5, 6, ribs; - - -, (broken line), border of lung.

The fact that on opening the abdomen the diaphragm remains arched, and that the lung by means of the tracheal clamp retains its position and does not collapse, warrants us in assuming that we can fairly judge of the position of the fluid during life by this method of investigation, especially as the contractility and distensibility of the lung appear to be perfectly retained after death, excepting in very cold weather, when it was found necessary to warm the cadaver. It may be objected to these experiments that the fluid was introduced at the bottom of the pericardial sac, while during life it might originate at the base of the heart. The fluid was therefore in several cases introduced where the pericardium is reflected over the great vessels; but even when it was in very small amount and quite insufficient to cause any increase of percussion-flatness it immediately ran down the side of the heart to the bottom of the pericardium. Even if it could be mechanically retained at the base of the heart, which was accomplished by inverting the cadaver, the resulting cast always had its broadest part towards the diaphragm.

The following cases are interesting and worthy of record, as illustrating the symptomatology of the acute pericarditis of infancy and childhood in its different forms. Primary pericarditis may be, as is now generally acknowledged, idiopathic or traumatic, and Hunter's case, as reported below, was supposed to be idiopathic with effusion and resulting in adherent pericardium.

Fig. 5.



The Lungs have been removed (Rotch).— ▨ A, normal shape of the heart in its pericardium; ▥ B, liver; ▤ D, effusion; A + D, the shape which the pericardium assumed in a case where considerable fluid had been introduced into the sac; S, sternum; ◎, nipple; 1, 2, 3, 4, 5, 6, ribs.

May 27, a girl ten years of age, previously healthy, and with no hereditary tendency to disease, complained of slight pericardial pain, increased somewhat on pressure; pulse 90 and regular; breathing rather hurried. No history of cold or injury. No affection of the joints, and no evidence of any other disease, local or general, the cardiac pain being her only complaint. A superficial, harsh grating was heard in the cardiac region accompanying both the systole and diastole of the heart and not affected by cessation of breathing. No endocardial murmur was heard, and there was no increase of the cardiac dulness. May 29, the area of cardiac dulness was much increased in every direction, and the friction-sound not so distinct. May 31, dulness still further increased, some cough, and considerable distress and oppression in breathing, pulse 120 and regular. June 1, physical examination gave the following results: the dulness extended from the second to the seventh left interspace and one inch beyond the left mammary line, extending also to the right a little beyond the median line of the sternum, when the patient was in the dorsal position, and about three centimetres to the right of the sternum when the patient was lying on the right side. An undulatory wave was seen with each cardiac impulse in the interspaces between the second and third and third and fourth ribs. The breathing was exaggerated over the right front, and at the right base behind there was enfeebled respiratory murmur and comparative dulness on percussion. Slight cough; urine scanty and not albuminous. The apex-beat, so far as it could be localized, was tilted upward and to the right. The dulness then began to decrease and the friction-sound to increase and then decrease until June 17, when they had both disappeared, and the patient was sitting up in bed feeling much better. July 25, the patient in the mean time being up and about, the heart-sounds were found to be normal, but the pulsation was slightly irregular. The area of dulness was normal. The apex-beat was in the normal position and was rather feeble. A retraction during the heart's systole was noticed in the third, fourth, and fifth interspaces, not noticeably affecting the lower half of the sternum, and remaining visible during deep inspiration.

Kerchensteiner has reported a case of idiopathic pericarditis in a girl eleven months old.

The next case is of great interest, not only as resulting from an unusual trauma, but also as suggesting the strong probability of the heart's having been punctured. It is reported by Dr. W. F. Morrison, of Providence.

A boy seven and a half years old fell on the sharp point of a slate-pencil which he was holding in his hand: the pencil penetrated the fourth left intercostal space close to the sternum. He was assisted to his feet, and breathed with difficulty: attempts to extract the pencil, made by his father with a pair of pliers, resulted in breaking off the pencil nearly even with the boy's body.

When seen somewhat later by Dr. Morrison, the boy was lying on his back, his arms raised, his face pale and anxious. He was breathing with considerable difficulty, and was very apprehensive of dying. The pulse was small and quick. The fragment projected only one-quarter of an inch, and could not be withdrawn until ether was given and incisions made above and below it, when, by firm, steady traction, the pencil, as shown in Fig. 6, was drawn from the chest. Blood, largely diluted with serum, welled from the wound, and later clear serum came to the surface. After the pencil was withdrawn, the boy took a few inspirations, when his limbs stiffened, he turned deadly pale and stopped breathing. There was divergent strabismus, and no pulsation could be detected in the wrist or carotids. Artificial respiration was made for two or three minutes, when he caught his breath and breathed again. The pulse was then 40 beats to the minute, and remained about 50 for an hour.

FIG. 6.

The wound was closed with two stitches and a cold-water compress applied: later in the day (four P.M.) the pulse was 140, the respirations 36, the temperature 101° F., and he had vomited twice. Six hours later the area of cardiac dulness was increased, the heart-sounds were muffled, and the respiration, which was painful and superficial, was found to be louder and harsher at the left apex than at the right. During the night the boy was restless and cyanotic, and in the morning his pulse was 156, respiration 44, and temperature 101° F. There was also great tympanites and episgastric tenderness. The urine contained no albumen, but a large amount of alkaline phosphates, and had a specific gravity of 1015. At noon on this day the percussion-flatness extended to the right of the sternum nearly to the nipple and upward to the second intercostal space.

During the next ten days the physical signs were those of pericarditis with effusion, the sounds disappearing when the pericardial effusion was the greatest and returning when absorption occurred. On the twelfth day after the injury the temperature was normal, the pulse 120, the respirations 35, and the effusion much less. The heart-sounds were correspondingly more distinct.

Dr. Thomas Dwight, Professor of Anatomy in the Harvard Medical School, who examined the boy on the fifteenth day from the time of the accident, gave as his opinion, from an anatomical stand-point, that the heart itself had been wounded; and Dr. Morrison concludes that this must have been the case, from the pencil entering the thorax three and one-half inches vertically, and directly over the right ventricle, from the great shock following the extraction of the pencil, and from the slow cardiac pulsations lasting more than an hour.

A case reported by Ashby illustrates a purulent pericardial effusion as it occurs in infancy, and shows the possibility of a spontaneous opening.

A wasted, fretful infant, four and one-half months old, was presented for treatment for a small, fluctuating swelling the size of a walnut and situated at the tip of the ensiform cartilage. The illness had lasted for some weeks, and was reported to have presented as symptoms feverishness and dyspnœa. The mother supposed that the infant was suffering from the results of a vaccination. There was dulness over the sternum. The abscess was opened, and pus escaped and continued to drain for a few days, when death took place rather suddenly. The post-mortem revealed the fact that the abscess communicated with the pericardium, which contained about one ounce of pus.

Somewhat similar cases have been reported where a purulent pericarditis has discharged under the left clavicle and in the second right intercostal space.

Chronic pericarditis may occur in infancy and childhood, as in adult life, but in its symptomatology has nothing distinctive of either age.

Diagnosis.—From what has been said above regarding the latency of the general symptoms in childhood and the difficulty of interpreting the local symptoms, it will be readily understood how important it is to investigate all the organs in a sick child, and thus by a process of elimination we are often enabled to make a differential diagnosis in the more difficult cases of pericarditis, by having our attention directed to the pericardium as a possible cause. Instances of this difficulty are seen in those cases where a distended pericardium has been mistaken for a left-sided empyema, reported by Ashby, Labric, and others. The condition, however, which most closely simulates a pericardial effusion, both in its general symptoms and in its physical signs, is the dilated heart.

A noted instance of this was reported to the writer by an interne of the Hôpital des Enfants Malades, where a little girl five years old, in the service of Dr. Henri Roger, presented all the signs of an abundant pericardial effusion. The case was under observation for several weeks, and Dr. Roger repeatedly marked out the area of dulness in his usual minutely careful way, and designated the exact spot where he intended to insert the trocar. In deference, however, to the opinion of his colleague Dr. Labric, who on general principles opposed the operation, the puncture was deferred; and finally the child died. The autopsy disclosed no effusion, but an enormously dilated heart. So closely, then, were the signs of a copious pericardial effusion simulated, in this case, by a dilated heart, as to deceive one so skilful as Dr. Roger, fortified though he was by a nineteen years' hospital experience, and considered a virtuoso in the art of physical examination.

Of all the physical signs of pericarditis, the friction-sound, when present, is most distinctive. Where, however, an effusion takes place,—which, as above stated, is especially frequent in children,—the friction-sound may not be found; and, as the heart's impulse may, as explained mechanically above, be clearly perceptible in a child with a considerable effusion, we are forced, by the similarity which at times arises between the general symptoms, the inspection, palpation, and auscultation, of a dilated heart and a pericardial effusion, to resort to the knowledge which we have obtained from, and the percussion-area of dulness which we have deduced from, the experiments mentioned above, which, although exact only for typical cases, will also prove of great aid in a very large number of cases. It will be necessary here to consider the possible area of dulness which may be produced by an enlarged heart, and, by comparing this area with what we have shown to exist in pericardial effusion, determine the differential percussion-signs of the two diseases. Owing to want of space, the writer cannot here introduce the results of his own observations, as well as those of others, on the area of absolute dulness of an extremely enlarged heart; but he has found that, although the relative dulness may extend to the right of the sternum from the second to the sixth rib, and perhaps to the distance of three or four centimetres on a level with the fourth rib, yet it would be rare to find this relative dulness invading the fifth right interspace more than two or three centimetres, and still more rare for the absolute dulness to be found in the fifth interspace at all, and even in the fourth interspace for more than one or two centimetres.

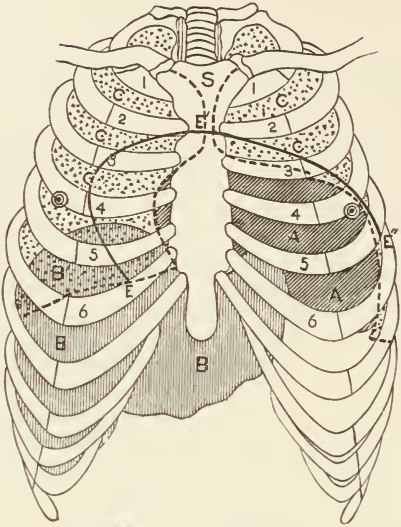
Now, on referring to Fig. 2 we find that these rules are exactly reversed in a pericardial effusion, for even a small amount of fluid finds its way to and produces absolute dulness in the fifth right interspace. (The writer, in one of his adult experiments, found that absolute dulness could be detected in the fifth right interspace when only from seventy to eighty cubic centimetres of fluid had entered the pericardium, which is from twenty to thirty cubic centimetres less than the amount reported by clinical observers as being the smallest which it was possible to make a diagnosis by.) The area of absolute dulness to the left of the sternum corresponds so closely, in its shape and extent, in enlarged heart, and in effusion, that for purposes of diagnosis it is of little use.

The writer wishes it to be understood that these deductions are held by him to be valuable merely as a working basis for future clinical investigation. He believes, however, that it will be found, where the distinction is to be made between an enlarged heart and a pericardial effusion, that absolute dulness of any considerable extent in the fifth right interspace means effusion, provided that other complications outside of the heart and pericardium can be excluded.

Fig. 7 represents the combined views of authorities on enlarged-heart dulness, and will be useful to refer to when we consider the question of paracentesis.

The following cases, taken from a number which have clinically come under the writer's observation, illustrate the difficulty of differential diagnosis between cardiac and pericardial disease where, as at times happens, we fail to find a friction-sound or murmurs.

FIG. 7.



ENLARGED HEART (Rotch).—▨ A, area of percussion-flatness caused by an enlarged heart: ▥ B, liver; ▦ B′, that portion of the liver which is covered by the right lung; ▩ C, lung; E E′ E″, the line marking the area of relative dulness of the enlarged heart; S, sternum; ◎, nipple; 1, 2, 3, 4, 5, 6, ribs; - - - (broken line), border of lung.

CASE I. Endocarditis; Dilated Heart.	CASE II. Pericarditis; Effusion.	CASE III. Endocarditis; Enlarged Heart; Pericardial Effusion.
Girl, eleven years.	Boy, six years.	Girl, eight years. August 3, 1887.
Attack followed acute articular rheumatism.	Attack followed acute articular rheumatism.	Attack followed acute articular rheumatism.
Orthopnœa; præcordial pain.	Orthopnœa; præcordial pain.	Orthopnœa; præcordial pain.
Heart's impulse feeble, but perceptible a little to left and below left nipple, fifth interspace.	Heart's impulse feeble, but perceptible a little to left and below left nipple, fifth interspace.	Heart's impulse feeble, but perceptible and felt all over cardiac area, with apex-beat a little below and to left of left nipple, fifth interspace.

CASE I. Endocarditis; Dilated Heart.	CASE II. Pericarditis; Effusion.	CASE III. Endocarditis; Enlarged Heart; Pericardial Effusion.
Vertical absolute dulness not increased.	Vertical absolute dulness not increased.	Vertical absolute dulness not increased.
Absolute dulness under the sternum and to the left of sternum; identical with Cases II. and III.	Absolute dulness under the sternum and to left of sternum; identical with Cases I. and III.	Absolute dulness under the sternum and to left of sternum; identical with Cases I. and II.
Absolute dulness did not extend to right of sternum.	Absolute dulness in fifth right interspace two or three centimetres from edge of sternum.	Absolute dulness in fifth right interspace three or four centimetres from edge of sternum.
Systolic murmur at apex.	Pericardial friction-rub at base.	Soft systolic murmur at apex, transmitted to axilla. Pericardial friction-rub at base.
Recovery.	Recovery.	August 6: Less dulness in fifth right interspace; apex-murmur much louder and harsh. August 11: Dulness only to right edge of sternum. August 18: Dulness only to middle of sternum; friction-rub ceased. December 1, 1887: Physical examination the same as on August 18, showing enlarged heart and mitral systolic murmur.

It will be observed that the symptomatology, both general and local, of these cases was (with the exception of the friction-sounds, murmurs, and percussion) identical, and that where an effusion was present dulness was found in the fifth right interspace, while where it was absent dulness was not found in this interspace. These especial cases with friction-sounds and murmurs were merely chosen so that there should be no doubt as to the disease with which we were dealing when testing the value of the percussion-limits for diagnosis.

The occurrence of pericarditis, with its accompanying effusion, has been referred to as liable to appear in the later stages of scarlet fever. Dilatation of the heart also occurs, according to Steffen, late in scarlet fever, especially where from the age of the patient (three to eight years, as shown by Gerhardt) the so-called physiological hypertrophy of the heart is present, and the tendency to enlargement is still further prompted by the increased blood-pressure from the diseased kidney. In addition to these inciting causes at from three to eight years, we find the fifth year[1] an especially critical period for the heart, as it is at this age that a comparative standstill in its growth takes place (Boyd) and yet its work goes on increasing. The age of the patient when from three to eight years should, then, be taken into consideration when we find a diminished resonance over the lower two-thirds of the sternum where in infants and adults we normally find resonance, since we may mistake a physiological area of dulness for the dulness of a small effusion. The writer has personally verified, by the percussion of a large number of subjects in the first twelve years of life, the following points. In infancy there is, as a rule, resonance under the sternum corresponding to the resonance of the young adult and the adult. From two to three years up to eight or ten years quite a number of apparently healthy, well-formed subjects were found to have dulness along the lower two-thirds of the sternum. It is well to state here that the writer has percussed very large numbers of infants and children, both healthy and diseased, and of all ages, in the fifth right interspace, and has found this interspace resonant.

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[1] A. Jacobi, Heart and Blood-Vessels in the Young.

In connection with pericardial effusions we should refer to the possibility of both complete and partial obliteration of the pericardial cavity occurring in children. Where severe cardiac symptoms are present and no valvular murmurs, we should, in youthful subjects, think first of degeneration of the heart-muscle itself, and next of pericardial adhesions. When, again, the absolute area of dulness remains unchanged and there are well-marked systolic retractions, the presence of pericardial adhesions is highly probable.

Prognosis.—In early infancy diffuse pericarditis is a very dangerous disease and usually leads to speedy death. In later childhood its course and results are subject to the same influences as in adults, and in the acute form the disease has a tendency to recovery.

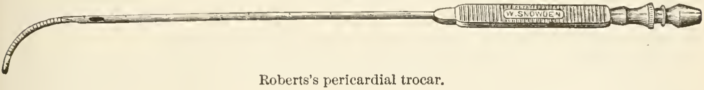
Treatment.—The treatment of pericarditis in the young does not differ materially from that of older subjects, and depends upon the various causes which have been spoken of in the section on the etiology of the disease.

The tendency to heart-failure, however, which is so pronounced in the child, should early in the disease be appreciated, and, while measures to relieve the affected organ should be adopted, as in the adult,—whether they be by means of absolute physical and mental rest, or with the rubber-tubing coil containing ice-water, or with the bromides and opium or salicylate of sodium,—we should, by the judicious use of digitalis, endeavor to forestall the crippling of the heart which is likely to occur after the early days of the disease. We should also make use of stimulants whenever there is a general indication for them.

Paracentesis of the pericardium is valuable in the child as in the adult, and should unhesitatingly be performed, no matter what the cause of the disease may be, whenever life is endangered from undue distention of the pericardial sac.

The description of the operation of paracentesis has been so ably presented by Dr. Roberts, in his work on "Paracentesis of the Pericardium," that it would hardly be well to repeat it here. Roberts's aspirating-needle, containing a flexible tube, which can be thrust forward so as to protect the heart from the point of the needle after the pericardium has been entered, can be used in serous effusions and where it is decided to tap to the left of the sternum. It is represented in Fig. 8. Roberts considers the space between the ensiform appendix and the left seventh cartilage the safest point for tapping. The fifth left interspace is, however, a favorite place with operators. The possibility of wounding the heart should be taken into consideration and avoided,—although cases have occurred where no harm has come from tapping the right ventricle. Deaths, however, are also recorded where the heart has been punctured in the region of the auricles and the upper segment of the right ventricle.

FIG. 8.



Roberts's pericardial trocar.

An important point, both in diagnosis and in treatment, should here be spoken of. It has been held, by certain authorities, that the heart's apex is found, in effusions, to be tilted upward and inward towards the sternal end of the fourth interspace,—that is, it is floated up by the effusion. Direct proof of this is, however, wanting, and it is believed by the writer, from his careful investigations on this subject, that this is an erroneous view. It would seemingly be impossible for the heart not to sink, rather than to be floated up, unless the specific gravity of the effusion was greater than 1050,[1] which it is highly improbable would occur in an ordinary pericardial effusion, for the specific gravity of a purely purulent fluid is only about 1032. How, then, can we explain the clinical phenomena of the heart-beat in the region of the fourth left interspace? Referring to Fig. 2, it seems plausible to account for this pulsation by the tumultuous action of that portion of the right ventricle which is seen to be free from the effusion in the fourth interspace when a small effusion is present.

On examining the cacao-butter casts, it is found, also, that this portion of the heart is, in the larger effusions, covered by a very thin layer of fluid, through which the impulse of the heart could easily be felt and seen. This fact is of especial significance when we consider that both Ludwig and Bowditch have observed that the impulse of the heart, as seen normally in the fifth left interspace, need not necessarily be caused by the heart's apex, but by a portion of the heart above the apex striking against the thoracic wall. We should here consider, also, that the impulse, in children, is often

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[1] The writer has determined, by direct experiment, that the specific gravity of a fluid must be 1050 in order that the heart should float in it.

normally in the fourth interspace. In Case III., described in the table on page 862, it is recorded that the impulse was felt throughout the whole cardiac area, but that it was still pronounced in the fifth interspace. Now, if in this case there had been a larger effusion, the apex and the lower segment of the right ventricle being surrounded by a mass of fluid, the impulse would have been lost in the fifth interspace, while in the fourth interspace, where the ventricle is covered by only a thin layer of overlying fluid, the impulse could have continued to be both seen and felt, thus simulating an apex-beat. The writer believes that this is the explanation of all these so-called misplaced apex-beats in pericardial effusions.

From the above facts,—namely, that the heart, when effusion is present, remains in its usual position, and does not, even when much enlarged, impinge on the *fifth right interspace,* and that the effusion, even when in as small an amount as one hundred cubic centimetres, is found in the *fifth right interspace,*—is it not more rational to choose the *fifth right interspace* as the point for tapping, thus avoiding all question of injuring the heart? When we tap the pleura, we avoid the heart as much as possible: why not carry out the same rule in paracentesis of the pericardium? The writer has tapped the pericardium in the *fifth right interspace* a number of times on the cadaver, and has removed the fluid as easily as in the fifth left interspace. So far as he knows, however, the pericardium has not yet been tapped in the *fifth right interspace* in the living subject: so that the practical bearing of the above remarks must be left for future investigation.

Hydropericardium, Hæmopericardium, and Pneumopericardium may all occur in childhood, but they appear to have no symptoms by which they can be distinguished from the adult disease.

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DISEASES OF THE BLOOD-VESSELS,

AND

THEIR OPERATIVE TREATMENT.

BY J. COLLINS WARREN, M.D.

ONE of the most marked of the congenital defects of the blood-vessels is stenosis of the aorta, or a constriction of that vessel in the neighborhood of the ductus arteriosus.

In early fœtal life the aortic arch terminates by a narrow funnel-shaped opening, known as the isthmus aortæ, in the centre of a second arch formed by the junction of the ductus arteriosus with the descending aorta. If the isthmus does not disappear with the termination of fœtal life, a deep constriction of the aortic wall is seen just below the opening of the left subclavian artery. The origin of this artery may be involved in the constriction, but this is not usual :[1] sometimes the constriction is below the opening of the ductus.[2]

The walls of the aorta are frequently healthy at this point ; in other cases, however, they are thickened and thrown into irregular folds, as if they partook somewhat of the anatomical character of the walls of the ductus.

Sometimes a complete obliteration of the ductus takes place during fœtal life, or there is an absence of the isthmus. The compensation for this narrowing takes place through an increased action of the left ventricle and a collateral circulation between the subclavian and the thoracic and abdominal aorta. There will also be a dilatation of the aortic area with endocarditis, and atheromatous changes and even aneurism may result.

The symptoms of this lesion are not usually pronounced in early life, but may show themselves in palpitations, dyspnœa, and the pulsations of the large collateral branches around the shoulders and the ribs.

Death may occur from rupture of the heart or aorta, from aneurism, from pulmonary complications, or from apoplexy.

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[1] Garland, Pepper's System of Medicine, vol. iii.

[2] Rauchfuss, Handbuch der Kinderkrankheiten, vol iv., Gerhardt.

This malformation is, however, fortunately exceedingly rare, although a slight degree of narrowing of the aorta is often seen at this point in new-born infants.

In the newly-born animal it has been found by experiment that the arterial pressure is very small: it is but ninety millimetres in the newly-born dog, whereas in the adult animal it amounts to one hundred and sixty or one hundred and eighty. Even as large arteries as the carotid are said not to spurt when divided.[1] It is partly due to this fact that the cord when not tied will often not bleed. The pulse of the infant and the pulse of the child differ greatly from that of the adult: the pulse of the fœtus varies from one hundred and twenty-four to one hundred and fifty beats; in the new-born child it is about one hundred and thirty-six in the average case; at six years it is one hundred, and at thirteen it is eighty-eight.

The pulse is less rapid in tall children than in short ones. It is often impossible to obtain the pulse in the usual way at the wrist, particularly during the first ten days of life; in which case the femoral or the carotid will be found more accessible. If the number of beats only is desired, the open fontanel offers the simplest means of observation. In healthy children the pulse should be regular and of equal strength. In young children there is frequently irregularity of the pulse due to momentary slight derangements of the digestion and other minor disturbance: with growth, however, this irregularity becomes less marked.

Sphygmographic study of the pulse shows an absence of dicrotism: this is due partly to the diminished tension and partly to the short arterial circuit.[2]

The strength of the pulse is easily impaired by slight functional disturbances.

The rate of growth of the different arteries varies considerably after birth. It is smallest in the carotid, and greatest in the renal and femoral arteries. These differences are due to the variation in the growth of the various parts of the body which the vessels supply.

Any variation in the normal rate of development may therefore have an important bearing on the nutrition of certain portions of the body or on the health of the individual.

All the large arteries continue to grow until the twentieth year, but do not entirely cease to develop after that period. In the veins numerous valves aid the circulation, both in the extremities and in the intestines,[3] which disappear in later life.

An insufficient growth of the heart and blood-vessels is often found. Either may occur alone. The aorta may not exceed in size the normal iliac or carotid: the heart of a child nine years of age may be no larger than that of a new-born infant. Rokitansky observed the deficiency particularly in females, and with it an imperfect development of the body and especially of the sexual organs.[1] Virchow gave to this condition the name of hypoplasia of the vessels. He showed the frequent concurrence of chlorosis, due to imperfect development of the arteries, and also of the hemorrhagic diathesis, which was regarded as caused by the increased blood-pressure and an incomplete development of the wall of the vessel. Other manifestations of disease are produced by this condition of the vessels. Anæmia associated with degeneration of the tissue of the heart, and endarteritis with fatty degeneration and thinness of the vessels, are not infrequent accompaniments. Jacobi[2] mentions the case of a lady, whose death occurred at thirty-two years of age with granular degeneration of the heart, in whom the large vessels were of uncommonly small circumference. Certain vascular districts only may be affected, as in the lungs or the kidneys, giving rise to strong predisposition to disease in these organs: all such troubles may become more manifest at the age of puberty, when, in the normal condition, an unusual development of the heart and aortic system takes place. Swelling and congestion of certain organs may occur, and also nutritive disorders such as are seen in the bones in rickets. Catarrh of the pharynx and respiratory organs may also be caused by the slowness of the circulation when the normal relation between the heart and the blood-vessels is disturbed. Apart from disease, the walls of the aorta may be unusually thin and the elastic coats more yielding. Many anomalies are also sometimes observed in the origin of the intercostal arteries, more frequently in the thoracic than in the abdominal aorta. A peculiar wavy and net-like condition of the intima was seen by Virchow, due to thickening of that tunic, giving rise to a predisposition to atheromatous changes in the walls and to the development of aneurism.

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[1] Jacobi, Heart and Blood-Vessels in the Young, Brooklyn Medical Journal, March, 1888.

[2] Keating and Edwards, Archives of Pediatrics, December, 1888.

[3] W. S. Bryant, Boston Med. and Surg. Journal, October 25, 1888.

Jacobi mentions the cases of two infants in which bleeding occurred spontaneously, merely from excessive thinness of the walls of the vessels. The blood would trickle from the surface of the lower extremities like perspiration, in drops, day after day, until the baby died of exhaustion. Many of these cases of hemorrhagic diathesis are, however, due to syphilis. Actual rupture of the aorta from thinness of the walls is reported by Rokitansky.

Imperfect development of the sexual organs and other portions of the body may be due to this condition.

The symptoms of this condition include anæmia, palpitations, and disturbances of the sexual functions especially in females.

In rapidly-growing children there will frequently be found a disproportion between the size of the body and the vigor and development of the arterial system. Disturbances of the circulation will be especially apparent at this period. There will be a disproportion in the force of the heart-beat and the strength of the pulse at the wrist. Palpitations are observed with unusual frequency at such times. Such conditions necessitate careful supervision of the child's daily life both at home and at school, and especial attention to the regulation of the diet.

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[1] Rauchfuss, op. cit.

[2] Op. cit. p. 12.

Omphalitis.—Inflammation of the tissues of the cord and navel are frequently observed in cases of premature birth. If the inflammation extends from the tissues which form the stump to the vessels, the condition may become a grave one. The complication begins as a periarteritis or a periphlebitis, and, when the inflammation extends through the coats to the interior, thrombosis will take place. Runge[1] considers arteritis a more dangerous and more frequent complication than phlebitis, contrary to the usual theory. Of fifty-five autopsies performed by him, in fifty-four arteritis was found, and in only one case did phlebitis exist. This great predisposition of the arteries to inflammation is due to the unusual thickness of the periadventitial tissue, which is nearly double that seen in the veins. When the tissue once becomes involved in the inflammatory process, the inflammation may creep along the walls of these vessels for a considerable distance into the anterior, but does not extend beyond the point at which the hypogastric arteries are reflected upon the walls of the bladder. This tissue is found to be infiltrated with exudation, and at points to contain foci of pus. The disease is septic in origin and usually terminates in a fatal septicæmia. A frequent complication is pneumonia, which was found in more than half the cases by Runge. The source of the infection is usually found in another child, as the disease generally occurs in hospitals, but it may come from the mother.

Phlebitis is a much less frequent form of inflammation. When once developed, it may extend along the umbilical vein to the liver and be followed by hepatitis. There will be a corresponding constitutional disturbance, and both the fever and the icterus will usually be observed.

The most dangerous form of hemorrhage which occurs in the early days of life is that which takes place from the umbilicus. Umbilical hemorrhage is of two kinds: that which takes place from the umbilical vessels, and that which consists in an oozing from the tissues of the navel.

The umbilical vessels do not usually bleed when cut, owing to the large amount of muscular fibre which they contain, which causes a powerful contraction of the cut ends. The expansion of the lungs also favors hæmostasis by lessening the pressure in the descending aorta and hypogastric arteries. If the lungs do not expand well, the cut vessels are much more inclined to bleed.

It is found[2] that the cessation of pulsation in the umbilical arteries begins at the placenta and gradually works towards the umbilicus. Even where these vessels have ceased to beat, the hypogastric arteries may still continue to pulsate strongly. If the cord is divided close to the navel, the danger of bleeding will therefore be greater. A hot bath may favor bleeding from these vessels by relaxing the constricting fibres. Mummification of the cord of course averts this danger, but if moist gangrene take place, as sometimes occurs, bleeding may result.

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[1] Runge, Die Krankheiten der ersten Lebenstage, 1885.

[2] Runge, op. cit.

Slight bleedings at the time of separation of the cord are easily controlled, and the forms of hemorrhage from the vessels here described are not specially dangerous to life. Removal of the clots and free exposure to the air may often be sufficient to arrest further bleeding. Firm compression by an antiseptic pad held in place by a broad strip of adhesive plaster encircling the body will probably also be efficacious.

A far more grave accident is the so-called idiopathic hemorrhage or bleeding from the capillaries of the stump of the navel. This consists in an oozing of blood, either before or after separation of the cord, from the navel, from no visible vessel. There has been much speculation as to the origin of this form of hemorrhage. Minot[1] regards it as one of the various manifestations of the hemorrhagic diathesis. Bleeding from the stomach and from the intestines is a not infrequent accompaniment. Many writers have, however, attributed the bleeding to the icterus which is so frequently seen with it. The presence of icterus has been ascribed to obstruction or absence of the gall-ducts, but it is more probably hæmatogenous and dependent upon a septic condition. The toxic effect of the bile or septic material is supposed to impair the blood, rendering it thinner and less coagulable. The pathology of this affection is more fully described in the article on diseases of the umbilicus.

The disease is a most fatal one, Grandidier placing the mortality as high as eighty-three per cent. Fortunately, it is extremely rare: Dr. Thayer[2] refers to 24,533 births with only five cases of hemorrhage.

Compression, styptics, transfixion with needles and ligature, and actual cautery have all been unsuccessfully employed. One writer has described this form of bleeding as fatal by virtue of necessity, and beyond the reach of medical agents.

Spontaneous aneurism in subjects under twenty years of age is an exceedingly rare affection. Intracranial aneurism appears to be the most common form; but aneurisms of the aorta and other arteries in the body are also reported.

Excluding the intracranial variety, R. W. Parker[3] has collected fifteen cases, being all he could find of the original records. In some of these, disease of the aortic valves existed: in two cases only was it stated that the arteries were diseased, and but in two cases was the heart stated to be healthy. He reports the case of a boy aged twelve with spontaneous femoral aneurism: old hip-disease existed in the other limb. The femoral was tied, and the wound healed over, but the child died twenty-five days later from epistaxis. At the autopsy disease of the aortic valves was found. Bryant and Goodhart[1] give a case of aneurism in a young subject, associated with infarctions in internal organs.

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[1] Amer. Jour. Med. Sci., 1852.

[2] N. Y. Med. Jour., Oct. 17, 1885.

[3] Medico-Chirurg. Trans., vol. lxvii., 1884.

Dr. Norman Moore[2] reports the case of a child seven years of age who died suddenly. There was a history of previous acute rheumatism. The heart was hypertrophied; growths were found on the mitral valve and also on the aortic valves. There was an aneurism, the size of a hazel-nut, of the right common iliac. There was no evidence in this case of the existence of arterial disease either in the form of endarteritis or in that of degeneration of the coats of the arteries.

A second case, reported by the same author, was a child five years of age who died of tubercular meningitis. Growths were found on all the cardiac valves, and above the valves there existed an aneurism of the arch of the aorta, on the shallow pouch of which there were several small endarterial growths. The aneurism in this case was, he thinks, due to an acute endarteritis contemporaneous with endocarditis. The former case he regards as of embolic origin.

W. W. Keen[3] reports two cases of aneurism. One, in a Swedish girl eighteen years of age, was an arterio-venous aneurism of the brachial, which was cured by ligature. The disease appeared shortly after a sunstroke. There were signs of an aneurism of the innominate also. The origin of the aneurism was attributed to a diseased state of the arterial walls. The second case reported was an aneurism of the interosseous artery of the hand of a child eight years old, appearing spontaneously, and disappearing also spontaneously seven or eight years later.

Dr. Keen has collected from literature eleven cases in addition to those of Dr. Parker, making twenty-eight in all. In two cases the aneurism was situated at the elbow; in another case there was an arterio-venous aneurism of the occipital artery and the right and left transverse sinuses. Curiously enough, in this case, also, the disease was apparently due to sunstroke. In three cases the aneurism was situated in the arch of the aorta, one of these existing in a still-born child. In a girl twenty years of age there were multiple aneurisms and multiple emboli in the vessels of the brain, trunk, and extremities. In one case there existed an aneurism of the ductus arteriosus in a child one month old, doubtless due to insufficient cicatrization of the aortic wall at the point of insertion of the ductus arteriosus. Two cases can be added to this list, making in all thirty. Thibierge reports a case of aneurism of the arch of the aorta in a girl of seventeen.[4] Madrazo[5] mentions a case of popliteal aneurism opening into the knee-joint in a boy fifteen years old.

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[1] Trans. Path. Soc., vol. xxviii., 1877.

[2] Ibid., vol. xxxiv., 1883.

[3] Phila. Med. News, 1887.

[4] La France Méd., Paris, 1881, ii. 913.

[5] L'Echo Méd., Toulouse, 1888, 2e sér., ii. 93.

The frequent association of aneurism with heart-disease at this period of life, and the rarity of arterial degeneration, make it highly probable that there is a close connection between these two affections.

The origin of aneurism from embolism was, we believe, first pointed out by Ponfick; but this view has been frequently confirmed by English writers.

Precisely how the embolus gives rise to the local changes which terminate in dilatation of the vessel is not clear in all cases. It is possible that the embolus may be arrested in an artery which is gradually being occluded by endarteritis, and that the remainder of the softened area is dilated by the force of the current. Goodhart publishes cases of aneurism following ulcerative endocarditis, and attributes the dilatation of the artery to a virulent action of the embolus which led to changes in the walls of the vessel at the point of obstruction. Parker doubts the embolic origin of these aneurisms, and attributes the dilatation to local changes similar to those which produced the emboli.

In writing on cerebral aneurism, Kidd[1] remarks, "The dilatation consequent on the partial obstruction causing interference in the nutrition of the contiguous parts as well as of the walls of the artery itself, the weakened arterial wall yields at the spot where it is least supported by the surrounding tissues, and gradually an aneurism is formed." He suggests that the sharp point of an embolus may occasionally pierce the wall of the vessel and thus give rise to aneurism.[2]

Intracranial aneurism is perhaps the most common variety of spontaneous aneurisms in children. Church publishes a table of thirteen cases in subjects under twenty years of age. In seven of these cases heart-disease existed, and in six of these there were vegetations upon the valves. He regards this form of aneurism as due to embolism, and is inclined to think that disease of the arterial wall is rarely if ever a cause of the disease. Yet West[3] reports a case of aneurism of the left middle cerebral artery in a boy twelve years old following scarlet fever at eight years of age. There was, indeed, mitral insufficiency. Keating and Edwards,[4] however, regard this as a case of dilatation due to atheromatous degeneration of the vessel. The same authors caution against mistakes in diagnosis of aneurism, which are by no means uncommon. Hare[5] reports a case of spurious aneurism of the innominate in a girl aged seventeen. In this case the most definite signs of true aneurism were present, yet a post-mortem examination proved the absence of any lesion of the blood-vessel.

Endarteritis with degenerative changes seems to be a rare affection in children. Judging from the cases cited, it would appear that the blood-vessels of the brain and the walls of the aorta are the points chiefly affected, although other large vessels are occasionally the seat of atheroma.

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[1] St. Barthol. Hosp. Rep., vol. xxii., 1886.

[2] Ibid., 1870.

[3] Path. Trans., vol. xxxii., 1881.

[4] Diseases of the Heart and Circulation, 1888.

[5] Med. News, Oct. 1, 1887, p. 388.

Traumatic aneurism is not of infrequent occurrence in childhood, owing to wounds inflicted by the jack-knife and other sharp-pointed instruments. It is said that wounds of the femoral artery are not infrequently caused during the operation of whittling, when, the knife being accidentally dropped, the child quickly closes his thighs to catch the falling object.

Such is the probable origin of a case reported by the writer.[1] The patient entered the Massachusetts Hospital at the age of twenty-five years with a large pulsating tumor on the middle of the left thigh. He had stabbed himself, while whittling, twelve years before: a fortnight after the accident a pulsating swelling was noticed the size of a pullet's egg. It increased very slowly in size until six months previous to his entrance to the hospital, since which time it had grown rapidly. It had two lobes, each about as large as a medium-sized cocoa-nut. It proved to be a varicose aneurism. The "old operatiou" of Antyllus was proposed, the vessels being tied at each end and the sac dissected out. The patient made a good recovery. An interesting feature of this case was the long duration of the aneurism.

The old rule, that in traumatic aneurism both ends of the vessel should be sought and ligatured, has not been changed. It is probable, however, that the method of Hunter, which consists in the application of a ligature to the proximal end of the vessel at a point of election, would prove as successful in traumatic as in idiopathic aneurisms in children.

The application of the Esmarch bandage to an aneurism does not seem well suited to this age, nor does the method of proximal pressure by the tourniquet. The ligature applied with antiseptic precautions seems a much more simple and efficient remedy. At an early age the dangers of gangrene of the limb, of sloughing of the sac, or of secondary hemorrhage are probably so slight that they may be disregarded in making a choice of operation.

The ligature of a large artery has become a simple and comparatively harmless operation at the present time. An incision having been made through the integuments, the sheath of the vessel is sought for and laid open directly over the artery. The vessel is then slightly freed from its lateral attachments to the sheath by the point of a director. A bent aneurism-needle is then passed between the vessel and its sheath from the side next the vein. Care should be taken to tie the first turn of the knot sufficiently tight, so that the lumen of the vessel shall be completely occluded and no blood flow through: when this has been done, it will be found that the inner and middle walls have been more or less completely ruptured, according to the size or the strength of the vessel.

There appears to be no special advantage in the rupture of the inner walls, nor is it desirable to place the ligature so that the walls shall be simply brought in contact without rupture. The latter manœuvre is, indeed, difficult to accomplish, for, if care be taken not to rupture the walls, the lumen of the vessel may not be completely occluded and blood may continue to flow.

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[1] The Lancet, April 28, 1883.

It has at different times been thought of great importance that a special kind of ligature should be applied. Different kinds of material have been substituted for silk, as catgut, tendon, iron and silver wire, etc. A broad flat ligature was used in old times. Two ligatures were sometimes placed close together or a slight distance apart, and the vessel cut between them, so that each end could retract within its sheath. All of these expedients were adopted to promote healing and lessen the danger of secondary hemorrhage. This can be accomplished by the favorable influences of aseptic conditions. All that is now required of the ligature is that it shall not introduce septic material into the wound, and that it shall be made of material strong enough to retain its hold upon the vessel.

The process of repair after ligature is briefly as follows. A certain amount of inflammation takes place around the knot as a centre, varying, of course, with the amount of injury inflicted by the operation or the septic influences at work. This produces a growth of inflammatory tissue about the point of ligature which covers in the two ends of the vessel. If examined at the end of a week, this new tissue, if the traumatism be severe, will form a spindle-shaped mass covering the ends of the vessel for a considerable distance above and below the point of ligature. If, on the other hand, the conditions are strictly aseptic, the new growth will form only a narrow ring around the vessel, just sufficient to cover in the ligature. This ring was probably mistaken by Lister for the organization of his catgut ligature.

Within the vessel the lumen is occluded by a thrombus. This also varies greatly with the amount of traumatism present. The proximal is usually the larger of the two, and may extend to the origin of a large branch. If aseptic conditions have been preserved in the application of the ligature, the thrombosis will be slight. It has been maintained that under these circumstances the coagulation of the blood will not take place. It is doubtful, however, whether an artery ever heals without a certain amount of thrombus-formation.

As stated in another article,[1] thrombosis accompanies occasionally the closure of the ductus arteriosus, and it is, of course, the rule in the healing of the hypogastric arteries.

The ligature, when properly applied, divides the intima and a greater portion of the mediæ, and holds the fibres of the adventitia firmly pressed together in a tendon-like mass. The granulation-tissue formed about the knot gradually softens down the fibres of the adventitia, and the band which still holds the two ends of the vessel is thus divided, and a gradual separation of them takes place, leaving the ligature, midway between the ends of the vessel, embedded in the newly-formed tissue or callus.

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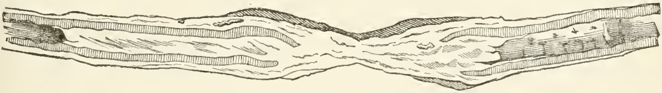
[1] Vol. i. page 254.

The ends of the vessel, now liberated from the ligature, unfold, and the granulation-tissue makes its way into the interior of the vessel, growing into the thrombi. Vessels accompany this tissue, and, as the thrombi are absorbed, a communication is established between them and the lumen of the vessel. The external and internal calluses are now gradually absorbed, cicatricial tissue taking their place. The ends of the vessels are then held together by a ligamentous band, which separates the remains of the external callus. Their walls are still slightly separated, but the space between them is filled out by a cicatricial tissue. This is composed internally of an endothelium, beneath which is found a layer of newly-formed muscular tissue composed of long spindle-shaped cells with staff-shaped nuclei: beneath this is a connective-tissue layer. We have thus represented in the cicatricial tissue the three coats of the vessel. The shape of this cicatrix varies considerably. In vessels which have no large branch at or near the point of ligature, it is crescentic, the two horns running up symmetrically on each side of the vessel. The accompanying drawing is taken from the carotid artery of a horse four months after ligature. The growth has extended a considerable distance into the interior of the divided extremities of the vessel. The external callus is in the process of absorption.



Carotid artery of a horse four months after ligature.

If a large branch is given off near the point of cicatrization, the new tissue is strongly developed on the opposite side of the vessel, and the other horn of the crescent terminates at the point of bifurcation.

A small artery is usually seen in the centre of the cicatrix, leaving the vessel at this point and terminating in a fine capillary net-work which ramifies in the ligament uniting the two ends.

It will thus be seen that the process of repair is a prolonged one, and that the vessel is first sealed by a provisional tissue which ultimately gives place to the permanent cicatrix. In large arteries this process is not fully completed until the end of three months from the time of ligature. The process is not unlike that which takes place in bone, and is what one would expect to find in complicated structures in which histological changes take place slowly.

The nature of the permanent cicatrix is such that it is well calculated to withstand the arterial pressure, and, when allowed to complete its formation, no aneurismal dilatation takes place. If a large artery be wounded by puncture, the circulation will usually be re-established before the repair is complete, and aneurism is therefore a common result of such injuries.

In amputation-stumps the process of repair differs materially from that which is seen after ligature in continuity. A considerable contraction of the main vessel takes place throughout its whole length, and there is a compensatory endarteritis to accommodate still further the lumen to the diminished vascular district which it nourishes. The vascular supply is therefore carried through a system which gradually breaks up into numerous branches, distributing the blood equally to all parts of the end of the stump.

HÆMOPHILIA.

By THOMAS D. DUNN, M.D.

Definition.—A congenital and usually hereditary vice of constitution, characterized by a hemorrhagic diathesis, and associated with a tendency to swelling of the joints.

Synonymes.—Hereditary hemorrhage, Hemorrhagic diathesis, Idiosyncrasia hæmorrhagica, Hæmatophilia; French, Hémophilie; German, Bluterkrankheit. The Germans call a sufferer from the affection "bluter," which corresponds to our word "bleeder."

This article will consider the congenital and hereditary affection, and not the transient hemorrhagic diathesis seen in certain diseases,—*e.g.*, scurvy, purpura simplex, anæmia, and purpura hæmorrhagica.

History.—The earliest historical mention of true cases of habitual hemorrhagic diathesis is found in the writings of an Arabian physician who died at Cordova A.D. 1107. Alexander Benedictus relates the case of a Venetian barber who bled to death from a wound of the nose caused by clipping the hairs. Virchow[1] calls attention to a case described by Hochstetter in 1674. Legg[2] discovered an authentic case reported in the "Philosophical Transactions" in 1743. Fordyce[3] described a Northamptonshire family several members of which were bleeders. These reports, with those of two other briefly-mentioned cases in Germany in 1793 and 1798, constitute the literature of the subject at the beginning of the century.

American physicians were the first to recognize fully and describe the nature of the affection. Otto[4] describes a New England bleeder family in which the disease could be traced back nearly a hundred years. He also refers to three others observed by Rush and Boardley. He was the first to use the word "bleeder," and to note the immunity of females in families suffering from the disease, and their tendency to transmit the disposition. E. H. Smith[5] gives an account of a boy affected with it in 1794. In 1813,

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[1] Virchow's Archiv, Bd. xxviii.

[2] Hæmophilia, London, 1872.

[3] Fragmenta Chirurgica et Medica, London, 1784.

[4] Medical Repository, New York, 1803, vol. vi.

[5] Phila. Med. Museum, vol. i., 1805.

Hay[1] reported the Appleton-Swain families. In 1817 Buel[2] reported the Collins family, and in 1828 R. Coates[3] a Delaware County, Pennsylvania, family, several bleeder descendants of which have come under my observation. Notable cases have since been described in American journals by Hughes, Gould, Harris, Holton, Blake, Wendt, Caldwell, and myself. In England very few cases had been reported prior to the publication of Legg's valuable treatise in 1872.

In Germany important contributions to the literature of the disease were made by Nasse, Schönlein, Wachsmuth, Lange, Virchow, Immermann, and others. In 1855, Grandidier published his excellent monograph, of which a new edition has appeared. This contains exhaustive statistical research of all the reported cases. In France two important articles have appeared,—Gavoy's in 1861, and Simon's in 1874. These articles, with numerous cases and papers of value recorded in Transactions and journals, comprise the literature of the subject.

Etiology.—Of all known causes of the disease an hereditary family disposition is the most important. It may arise spontaneously in a child, but we are ignorant of the conditions under which it develops in healthy stock. A few cases have been alleged to originate from fright or fear, or from the intermarriage of relatives, the latter being urged on account of the frequent appearance of the affection among the Jews. The early age at which circumcision is performed among this people gives an excellent opportunity for the disease to manifest itself, but they are probably no more prone to the disease than other races. In cases supposed to have generated *de novo*, a careful study of the family history will often show an hereditary disposition to bleed. Among the poor it is difficult to get a history farther back than parents, rarely grandparents; and the affection frequently skips a generation, reappearing with its original severity. Grandidier says, "It is the most hereditary of diseases."

In two hundred and thirteen families six hundred and thirty-one were affected; these, with one hundred and twenty cases I have collected in thirty-seven families[4] and Immermann's nineteen unincluded cases in six families, give a total of seven hundred and seventy cases in two hundred and fifty-six families, or an average of more than three bleeders to a family.

Next to heredity, sex is an important factor of the disease, only eight per cent. of cases being females. Of sixty-four bleeder families, in five the sons and daughters were alike affected, in twenty-seven all the sons were bleeders, and in six of these there were no daughters. The mode of transmissson of the bleeder tendency is very curious and interesting. The daughters in bleeder families are *par excellence* conductors of the disease. They may themselves be healthy and marry healthy husbands, yet the habit is likely to be transmitted to their sons. The daughter of a bleeder family, herself a bleeder, is no more liable to transmit the disposition than her non-bleeder sister. A son of bleeder stock, himself a bleeder, should he live to beget children, does not commonly transmit the tendency to his children, but it is sure to appear in his grandchildren through his daughters. Hay[1] describes a family in which the tendency was well marked for ninety-five years. Legg[2] exhibits a family tree in which it has existed two hundred years.

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[1] New England Med. Jour., 1813.

[2] Trans. Med. and Phys. Society of New York, 1807.

[3] North Amer. Med. and Surg. Jour., 1828.

[4] Amer. Jour. Med. Sciences, January, 1883.

Hæmophilia manifests itself at an early age. In ninety-five cases in Grandidier's tables it appeared in fifty-eight during the first year; in nine, during the second year; in eight, during the third; in two, during the fourth; in five, during the fifth; and in five, during the sixth year. In the cases I reported, thirty-four began to bleed before the eighth year, while only two were known to have commenced after the eighth. It rarely manifests itself for the first time after the twelfth year.

The older writers attached importance to the constitution and temperament of bleeder children, but more recent authorities do not recognize such peculiarities. Wachsmuth and Grandidier described two forms,—erethetic and atonic. Unusual mental activity has been ascribed to bleeder children, which can be accounted for by the studious habits of the sedentary life entailed, rather than by greater intellect.

It is chiefly distributed among the Anglo-Germanic races. Of two hundred and nineteen families, Germany furnishes ninety-four, Great Britain fifty-two, North America twenty-three, France twenty-two, and other European countries the remainder.

It may occur in all conditions of life. Cold, damp, changeable weather often determines attacks, while a warm, equable climate diminishes the tendency. All writers have observed the great fertility of bleeder families. In families where all the boys are not victims, the first-born are less liable to bleed.

Pathology and Pathological Anatomy.—Not many morbid conditions aside from those of anæmia have been found in autopsies of bleeders with any degree of constancy. Blaydon[3] calls attention to the thinness and transparency of the vascular walls. Legg[4] reports a case in which Klein after a careful examination met with negative results. The superficial arteries, according to Immermann,[5] are large in proportion to those coming from the heart. Winters[6] has noted a similar condition, and that they were inelastic, resembling veins. Kidd[7] found proliferation and swelling of endothelial cells, and diminution and degeneration of the muscular coat of arteries and veins. The swelling of the joints, according to Dubois,[1] is due to extravasation of blood into the connective tissue surrounding the joints and into their synovial cavities. Later writers (Reinert, Assmann, Paget) confirm his observations. Osler[2] thinks "two circumstances combine in hæmophilia,—congenital fragibility of the vessels and a defect in the coagulability of the blood; but whereon these depend we are as yet entirely ignorant." In the absence of information as to what these changes are, theorizing, in a practical work of this kind, is better omitted.

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[1] New England Journal of Med. and Surg., 1813, vol. ii. p. 221.

[2] St. Bartholomew's Hosp. Reports, 1881.

[3] Med.-Chir. Trans., 1817, p. 217.

[4] Lancet, October, 1884.

[5] Ziemssen, vol. xvii.

[6] Dublin Med. Jour., September, 1880.

[7] Medical Times and Gazette, May, 1878.

Symptoms.—The first indication of the existence of this constitutional vice is an uncontrollable spontaneous or traumatic hemorrhage. As previously stated, it appears at an early age. For convenience, the symptoms may be considered under the following divisions (Legg): external bleedings, spontaneous or traumatic; interstitial bleedings,—petechiæ and ecchymoses; and the joint-affections.

Legg has pointed out three grades of the disease. The aggravated form is characterized by bleedings of every kind, external and internal, and the swelling of the joints: this form is seen in boys, generally lasts through life, and is often the cause of death. The intermediate has no tendency to joint-affections or traumatic bleedings, but there are frequent spontaneous hemorrhages from mucous surfaces and submucous ecchymoses: this form is most often seen in girls, and usually disappears at puberty. The third and lowest degree appears only in girls of bleeder families, and manifests itself in ecchymoses and in early and prolonged menstruation.

The prodromic symptoms of spontaneous bleeding are plethora, ruby lips, hot skin, headache, and irritability of temper. The bleeding may take place from the skin, from the mucous and sometimes from serous membranes. It may be preceded by pain over the body or in the joints. The capricious appetite of chlorosis is sometimes present. In young children the most commonly affected localities are the nose, tongue, and gums; next in order are the stomach, bowels, bladder, lungs, and kidneys. The quantity of blood lost varies from a few drops to enough to endanger life, the most frequently fatal bleeding being epistaxis. Hemorrhages from injuries and operations are in no way proportionate to the extent of the wound. Fatal bleedings have followed blows on the head, bites of the tongue, leeching, blisters, venesection, vaccination, circumcision, phimosis, extraction of teeth, and many other trivial accidents and operations. The hemorrhage is always a capillary oozing, which may be intermittent or continuous to exhaustion and followed by death in a few hours or after several days. Coates's case lost two quarts of blood in twenty-four hours, and it was estimated that the loss was three gallons in ten days. Wounds in bleeders usually heal rapidly, and often without suppuration. The condition after a severe hemorrhage is, of course, that of profound anæmia, from which the patient commonly recovers with remarkable rapidity. Interstitial hemorrhages—petechiæ and ecchymoses—are quite common, and may be subcutaneous, submucous, rarely subserous. They may be the result of slight injuries or spontaneous in their origin, may vary from the size of a pin-head to that of the hand, and display a red, purple, or bluish-black color, and are not uncommonly the only evidence of the disease. Hæmatoma are usually the results of blows, and often terminate in extensive sloughing. Petechiæ are most frequently found on the extremities, and crops are sometimes accompanied by pain and swelling of the joints.

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[1] Gaz. Méd. de Paris, 1838.

[2] Amer. Syst. Med., vol. iii. p. 936.

The *arthritic* factor of hæmophilia is very important, often troublesome, and in some members of bleeder families is the only manifestation. This has led the laity and older practitioners to call the affection "the hemorrhagic and rheumatic habit." The following is the order of frequency with which the joints are involved: knee, hip, elbow, ankle, wrist, shoulder. They are swollen, tender to the touch, and there is often effusion into the synovial cavities. Motion causes pain; redness is commonly absent, temperature sometimes elevated. In cold, damp weather they are usually worse.

Bleeder children pass through the diseases of infancy like others. Whooping-cough often produces epistaxis and conjunctival ecchymosis. The blood after a severe hemorrhage is thin and watery, and the anæmia following is often attended by digestive disturbances. The blood of hæmophilists clots firmly and quickly, and is rich in corpuscles and fibrin. A state of plethora is claimed by some writers to precede a hemorrhage, and the tolerance bleeder children have for the loss of blood is adduced in support of this view.

Diagnosis.—The diagnosis of hæmophilia is not often difficult in male children, but the milder forms seen in females are often attended with some doubt. When a child has suffered from severe spontaneous or traumatic hemorrhages, associated with swelling of the joints, and a history of heredity is known, a diagnosis of the disease is easily made. Great caution is required in the second form of the affection unless an hereditary taint can be discovered. Epistaxis in boys is common, and should not be classed with it unless associated with other features of the disease. It should be distinguished from umbilical hemorrhage of the new-born, which is often dependent upon jaundice, syphilis, or mycosis (Weigert[1]). Children of bleeder families rarely bleed from the umbilicus.

The following varieties of bleeding should not be confounded with hæmophilia: (1) Purpura simplex, seen often in badly-nourished and debilitated children. It commonly appears on the legs. (2) Peliosis rheumatica, a disease which resembles hæmophilia in the swelling of the joints and large interstitial hemorrhages. It may manifest itself in several members of a family. (3) Purpura of infectious diseases,—small-pox, scarlet fever, measles, cerebro-spinal meningitis, etc. (4) Purpura hæmorrhagica. This serious disease is characterized by extensive subcutaneous and submucous ecchymoses, but it is not infective nor dependent on any local disease. (5) Scurvy. The transient and acquired condition distinguishes the bleedings from those of hæmophilia. (6) Simple hemorrhagic diathesis, a tendency to uncontrollable hemorrhage from slight wounds without an hereditary family disposition. (7) Hemorrhagic sweating, seen usually in hysterical or epileptic females.

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[1] Cohnheim's Pathologie, vol. i. p. 382.

Prognosis.—While it is exceptional for the first hemorrhage to prove fatal, the younger the child is, the worse is the outlook. The longer a bleeder lives, the more hope is there that the tendency may be outlasted. The disposition may remain latent for years, then reappear, and the sufferer die of hemorrhage after a long life. The prognosis is worse in boys than in girls. In girls menstruation may be early and excessive, but is unattended with special dangers. The following table, compiled by Grandidier, comprises two hundred and twelve deaths from hemorrhage, one hundred and ninety-seven of which were males and fifteen females. It shows the excessive mortality in early life.

Within 1st year	22 males,	7 females.
From 1 to 7 years	89 "	3 "
" 8 " 14 "	39 "	1 "
" 15 " 21 "	24 "	3 "
" 22 " 50 "	17 "	1 "
Over 50 "	6 "	

In the series of cases I reported in 1883, there were sixteen deaths, nine of which occurred before the eighth year.

Long-continued oozing from lacerated wounds, profuse epistaxis, interstitial bleeding, and hæmaturia are very unfavorable. Hemorrhages from injuries about the face, scalp, tongue, and gums are controlled with great difficulty. In the second and third grades the prognosis is less grave.

Treatment.—The prophylaxis involves a very important question,—the marriage of members of bleeder families. The marriage of daughters of bleeder families should not be permitted, whether they themselves have the tendency or not, as their male children are certain to be affected. The same objection may be urged against the marriage of male bleeders, though their children are less liable to bleed than those of their sisters, but the disposition is likely to break out in their grandsons. The tendency is less frequently transmitted by non-bleeder males than females.

Members of bleeder families, particularly boys, should be guarded against injuries, and all the resources of conservative surgery should be exhausted before operations involving the knife are resorted to. Extraction of teeth should be absolutely prohibited. Bleeders should seek occupations in which there is little risk of injury. All alcoholic stimulants should be interdicted. When prodromata exist, a saline aperient is said to be efficient in preventing an attack.

Bleeders meeting with wounds should have absolute rest. The wound should be cleansed, and compression judiciously tried; but it must be borne in mind that pressure with hæmophilists is liable to be followed by ecchymoses and sloughing. Failing in pressure over the wound, or, if in a favorable locality, over the artery, the various styptics may be employed. The actual cautery has been used with benefit when other means have failed. Hot and cold applications have each proved useful. For epistaxis ice, tannin, and alum may be tried before plugging the nasal cavities. To arrest hemorrhage from an alveolus, after the use of the cautery, it should be plugged with lint saturated with Monsel's solution of iron. Ranger,[1] in hemorrhage of bleeders after extraction of teeth, advises an impression of plaster of Paris, the jaw to be held in position by a bandage. When plethora and congestion precede spontaneous hemorrhage, some authors (Wachsmuth, Stromeyer, Legg, and others) hold that it should not be checked at once, but allowed to exercise a derivative influence. Assmann and Reinert, on the contrary, maintain that this form should be treated with the same energy as hemorrhage of traumatic origin.

Internally, ergot, opium, gallic acid, and digitalis have been employed with alleged benefit. Otto[2] had good results from large doses of sulphate of sodium, while Fordyce[3] recommends sulphate of magnesium. Legg derived benefit from thirty- to forty-minim doses of perchloride of iron during the intervals, claiming that from its use spontaneous hemorrhages were less frequent and more manageable. Venesection has been resorted to in several instances, but with doubtful advantage. Transfusion has been used without benefit.

During the intervals the bleeder should have plenty of fresh air, light and supporting diet, and iron and cod-liver oil until the health is restored. Exposure to cold and damp should be avoided, and the body should be well protected by warm clothing. A residence in the South during the winter is desirable.

The treatment of the swelling of the joints should consist of the ordinary surgical measures. To remove effusion counter-irritation should be employed, but with caution, as it has been followed by alarming hemorrhage and extensive sloughing. Tincture of iodine painted above and below the joint is a useful and safe measure. An immovable dressing of plaster of Paris and firm bandaging have been found beneficial in some cases.

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[1] St. Thomas's Hosp. Reports, vol. vi. p. 121.

[2] Loc. cit.

[3] Loc. cit.

DISEASES OF THE SPLEEN,

AND

THEIR OPERATIVE TREATMENT.

By B. A. WATSON, M.D.

ANATOMY.

The spleen is a single vascular organ found in mammalia, situated in man in the left hypochondriac region, and, since it possesses no excretory duct, is commonly classed as one of the ductless or blood glands. The outlines of the organ are irregular and somewhat variable, either smooth or lobulated, while its general form is oblong and elliptical, and it is placed nearly vertically in the body.

It may be furthermore described, for the convenience of study, as possessing two surfaces,—one, the external, convex and free, turned towards the left, the other, the internal, concave, and directed to the right. There are likewise two extremities, the upper being thick and rounded, and connected with the diaphragm by a fold of peritoneum,—the suspensory ligament; the lower end is pointed, and in relation with the left extremity of the transverse arch of the colon.

The external or convex surface is smooth, extends upward, downward, and backward, and is in relation with the under surface of the diaphragm, which separates it from the ninth, tenth, eleventh, and twelfth ribs on the left side. The internal or concave surface is divided by a vertical fissure—the hilus of the spleen—into an anterior or larger and a posterior or smaller portion. The hilus is pierced by ten or twelve apertures, differing in size, for the entrance or exit of blood-vessels and nerves. The anterior portion of this concave surface of the spleen is in contact with the greater curvature of the stomach, especially when this viscus is distended; while the posterior portion of this surface covers the left kidney, the suprarenal capsule, and the tail of the pancreas.

The spleen is covered by two coats, of which the outer one is serous and the inner one fibro-elastic. The external serous coat is thin and smooth, and is derived from the peritoneum. The fibro-elastic coat is moderately strong, and forms the framework of the spleen. It invests the whole of the external surface of the organ, and is reflected inward on the vessels in the form of the sheaths, thus ramifying through every portion of the spleen and forming a complete net-work, commonly designated *trabeculæ.* It is this fibro-elastic material which allows the spleen to be distended and afterwards, under favorable circumstances, to resume its normal size. The meshes in this net-work are filled with a soft pulpy mass, of a dark reddish-brown color, consisting of colorless and colored elements.

In weight and dimensions the spleen varies more than any other organ in the body, a variation which depends principally on the amount of blood which it contains. The weight of this organ in a healthy person gradually increases from infancy to the age of forty, and then gradually decreases to extreme old age. The weight in the healthy adult male is from five to seven ounces, while in the female it is something less. The dimensions are generally as follows: length, five to five and one-half inches; width, three to four inches; thickness, one to one and one-half inches. The consistence of the spleen in a healthy state, so far as hardness is concerned, is about the same as that of the liver, and like that viscus it is friable.

The color in infancy or childhood is red or reddish, in the adult generally a grayish white, in old age commonly brown; although it should be remembered that the shades of coloring are more or less modified by the amount of blood the organ may contain at the time of the examination.

The arterial supply of blood to the spleen comes from the cœliac axis, through the splenic artery, which is remarkable for its relatively large size as well as its wonderful tortuosity throughout its entire length. The walls of this vessel are unusually thick; and its muscular fibres are uncommonly strong and elastic. It proceeds transversely to the left from its origin to the spleen, accompanied by the splenic vein, which lies below it, having passed in its course behind the upper border of the pancreas, giving off to this organ several small branches, and on arriving in the neighborhood of the hilus gives off the gastro-epiploica sinistra, a large artery which runs from left to right along the great curvature of the stomach and anastomoses with the gastro-epiploica dextra, and finally the splenic artery divides into several branches, which enter the spleen at widely divergent points, while others, the vasa brevia, turn backward to the stomach. The vasa brevia, numbering from five to seven, arise in part from the trunk and in part from the branches of the splenic artery, and are distributed to the great curvature of the stomach.

The splenic veins are much larger and more numerous than the splenic arteries. The veins anastomose freely, and by their junction form from four to six branches, which emerge from the hilus and converge to meet in the formation of a single splenic vein, the largest branch of the vena porta. The splenic vein returns the blood from the spleen, the pancreas, the duodenum, a portion of the stomach, the omentum, the descending colon, and the rectum.

There are two ligaments which serve to hold the spleen in its proper position. The ligament which is called the gastro-splenic omentum is formed of two layers of peritoneum reflected from the spleen about the hilus upon the cul-de-sac of the stomach, containing between them the splenic vessels and nerves and the vasa brevia, thus connecting the spleen with the cardiac end of the stomach. The suspensory ligament is formed by a reflection of peritoneum, which serves to connect the upper thick and rounded end of the spleen with the diaphragm.

The splenic nerves arise from the solar plexus accompanying the splenic artery.

The lymphatic vessels of the spleen are very numerous, and consist of a superficial and a deep set, which pass through the lymphatic glands at the hilus and terminate in the thoracic duct.

PHYSIOLOGY.

Though much has been written on this subject, little is known of the functions performed by this organ. From anatomical peculiarities of the spleen, it is believed that it exercises an action on the composition of the blood, which is submitted to a true filtration through its net-work and pulp. The spleen, being at all times elastic and contractile, is capable of rapid variations in its volume under the influence of the pressure of the blood within the vessels, or of nervous excitation.

The blood-pressure mechanically distends the organ, while a nervous excitation acting on its fibro-elastic tissues produces a marked diminution in its volume. This contractility of the spleen has been demonstrated by Claude Bernard, Schiff, Tarchanoff, Bochefontaine, and J. Bulgak. It has further been shown that this irritation may be made to the central end of a centrifugal nerve or to the peripheral end of a centripetal nerve. The same effect may be produced by the direct irritation of the surface of the spleen, or even by the irritation of the integument over the organ. It may likewise be caused by a cold-water douche or by a hypodermic injection of quinine, strychnine, camphor, or eucalyptus; while the contrary effect is produced by curara.

Schönfeld first called attention to the fact that the volume and weight of the spleen are both markedly increased about five hours after each meal. The correctness of the observation has never been denied. The question which has presented itself for consideration in this connection is, whether this condition arises from the part which the spleen plays in digestion, or is merely the result of pressure from an increased amount of blood in the splenic vessels. Numerous experiments have been made for the purpose of showing that the spleen performs some part in digestion, but a careful examination of the reported results in these cases seems to justify the author in pronouncing the verdict "not proven," and this opinion is further strengthened by the fact that the removal of the spleen has not been followed by any disturbance in the functions of digestion or assimilation.

We are now brought to the consideration of the supposed change in the blood produced by its passage through the spleen. It has been claimed by some authorities that the spleen takes an active part in the generation of the white blood-corpuscles and at the same time assists the lymph-organs in the performance of their special functions. The fact that the lymphoid changes are so constant in cases of leukæmia has led some to think that the spleen possesses leucocytogenic functions. A careful examination of that portion of the statement which relates to the formation of the white blood-corpuscles has satisfied me that it rests mainly on the assumption that there are found a greater number of white globules in the blood of the splenic vein than in that of the splenic artery; but more recent investigators, among whom are Tarchanoff and Swaen, have declared that there is little difference between the number of white corpuscles in the splenic vein and the splenic artery. These authors have further shown that the dilatation of the spleen is accompanied, in man, by a diminution of the number of white corpuscles in the general circulation, which is supposed to be due to a passive accumulation of these globules in the spleen.

The opinion advanced by certain medical authors, that the functions of the spleen are in some way connected or identical with those of the lymphatic glands and other adenoid structures, is surely entitled to some thought. If these functions are identical, it would naturally follow that the existence of a disease which involved the lymphatic system would likewise manifest itself in the spleen. Observation has, however, shown us that there is frequently a complete want of harmony in the functional action of these organs. For instance, in a case of leukæmia there will be found a marked enlargement of the spleen, lymphatic glands, and other adenoid tissues, but in cases of scrofula the lymphatic glands are generally enlarged, while the spleen does not participate in the morbid process. The assertion, which was maintained by a limited number of observers, that a general enlargement of the lymphatic glands followed the removal of the spleen, has been denied, and this question seems to be settled negatively. We are now led to the conclusion that there is insufficient evidence on which to base the opinion that the essential function of the spleen is the fabrication of the white-blood corpuscles or that its physiological functions are identical with those of the lymphatic glands.

It has been suggested that the spleen is the centre of the fabrication of the red corpuscles. The partisans of this opinion declare that the white corpuscles are changed in this organ into the red, and offer in support of this view the assertion that the transformation-state has been observed in both the red and the white globules found in the spleen-pulp. Foster[1] says, "In the spleen we find, as Kölliker long since pointed out, large protoplasmic cells in which are included a number of red corpuscles; and these red corpuscles may be observed in various stages of apparent disintegration. It is probable, therefore, that the spleen is the grave of many of the red corpuscles."

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[1] A Text-Book of Physiology, 3d ed., p. 38 *et seq.*

We now find ourselves compelled to admit, after having made a careful examination of the literature of the whole subject, that the physiological action of the spleen has not yet been satisfactorily determined,—although there is at least a probability that it may perform the work of a reservoir or diverticulum during certain intervals between digestion, and that it may likewise, under other circumstances, where the blood has been driven from the surface of the body from any cause, producing engorgement of the visceral organs, play a highly important *rôle.* It is a well-known fact that in many fevers the spleen becomes intensely engorged, but, owing to its distensible power, suffers no injury on such occasions, while at the same time it relieves other visceral organs from a blood-pressure which might be injurious.

GENERAL PATHOLOGY.

The spleen is often affected by diseases which are rarely diagnosed on the living subject, but which serve to make the post-mortem examination particularly interesting. This must necessarily be the case in those organs in which the physiological functions are not fully understood, and when there are consequently no recognized pathognomonic signs of disease.

The post-mortem examinations show the following morbid conditions of this organ: (1) congestion or engorgement, (2) acute inflammation,—splenitis, perisplenitis, or abscess,—(3) subacute diffuse splenitis, or hypertrophy, (4) interstitial chronic splenitis, or cirrhosis, (5) gangrene, (6) amyloid infiltration, (7) pigmentary alterations, (8) embolisms and infarctions, (9) rupture, (10) wounds, (11) displacements, (12) tumors, (13) syphilis. Pathological examinations have shown that disease of the spleen may be entirely limited to this organ, or it may coexist with a similar morbid condition in the liver, the intestines, or the lymphatic glands, dependent on a constitutional cachexia which had its origin in an old contagious disease.

It may, however, happen that the post-mortem conditions observed will fail to show whether they were the cause or the result of the disease, owing to the fact that the physiological functions of the spleen are still very imperfectly understood: for example, the alterations in the blood undeniably accompanying certain affections of the spleen, and the cachectic state of the patient which indicates an evident dyscrasia; but what is the evidence which connects this and the blood-changes with the spleen as the specific result of the disease?

This question cannot be answered intelligently until we know exactly the action of the spleen on the blood. It is, however, thought that in cases of leukæmia and hydræmia the alterations in the blood may be reasonably attributed, at least in some instances, to a perversion of the functions of the spleen. The supporters of this opinion have maintained that any treatment which would remove or relieve the engorgement of this organ would ultimately result in the recovery of the patient. The recommendation for the extirpation of the spleen is based on this idea. The supposition that the removal of the spleen would modify the course of the disease in cases of leukæmia or malarial fever is certainly very plausible, but, nevertheless, unreasonable, since the removal of the local and primary affection would not restore the healthy functions of this organ, nor even necessarily abrogate the constitutional cachexia which is commonly coincident to the morbid process. Professional experience in the treatment of cancer and of many other allied conditions abundantly justifies this course of reasoning.

In all cases of purely splenic leukæmia there has been observed in the early stage of this disease more or less engorgement of the spleen, while in those cases commonly designated lymphatic leukæmia the enlargement commences in the lymph-glands and the spleen is only secondarily involved. The most characteristic pathological changes primarily observed are in the blood, and consist in a more or less marked relative increase of the white corpuscles, but, owing to the fact that the physiological functions of the spleen have not been determined, we are unable to give to this pathological condition a scientific interpretation. Should the number of the white corpuscles become relatively great, there will be observed a change in the color of the blood, which may assume a grayish red and resemble a mixture of blood and pus. This morbid condition of the blood is likewise associated with certain chemical changes whereby the water is increased and the solids of the fluid are diminished in quantity. It is presumed that the hemorrhagic diathesis which is a very frequent concomitant of leukæmia may be explained on the basis of the altered condition of the blood, and possibly some degenerative changes in the walls of the blood-vessels.

In regard to the truly splenic origin of dropsical affections, or those which can be clearly traced to a perversion of the functions of the spleen, there is certainly abundant reason for believing that these cases are extremely rare. Pathologists have frequently demonstrated the fact that many of these cases, which the clinician attributed solely to a perversion of the functions of the spleen, were really caused by lesions of the heart, liver, or kidneys. Nevertheless, it must be admitted that there are many cases of dropsical effusions, arising in connection with splenic lesions and an anæmic cachexia, in which the pathologist has thus far failed to discover any other cause to which the dropsy may be attributed than the existing morbid state; but here is met a most perplexing problem: what *rôle* is played by the splenic condition in the dyscrasia? or, *vice versa*, what *rôle* does the dyscrasia play in the splenic condition?

It is, however, a well-known fact that hypertrophy of the spleen may end in the compression of the inferior vena cava, and thus cause a mechanical œdema of the lower extremities. It may likewise obstruct the portal circulation, or, what is more frequent, it may be coincident with or even follow an analogous hepatic lesion, with production of ascites. Finally, an existing splenitis may excite a chronic peritonitis which may be followed by an intra-abdominal effusion.

GENERAL SEMEIOLOGY.

Disease of the spleen is indicated by the existence of more or less marked signs, such as an increase in volume of the organ or a change in its consistence, pain, etc. The augmentation in the volume of the organ is attended with an increase in its weight. The principal means relied on for determining the enlargement of the spleen are inspection of the abdominal walls, palpation, and percussion and auscultation, while it is necessary at the same time that the examiner should keep in mind the normal dimensions and situation of the organ. Inspection of the splenic region affords us invaluable aid in diagnosing hypersplenic conditions. We may discover by this method of examination the want of symmetry between the two hypochondriac regions. The left, which is normally depressed, may be raised by a sort of intra-abdominal cake, in which may be traced out the form of the spleen.

It is highly important, in all our examinations of the enlarged spleen, to keep in mind the fact that this organ preserves the same form and relation to other organs in disease as in health. An exception to this rule is, however, found in cases of so-called patulous spleen, which possesses great length and correspondingly less width. Consequently, the surgeon is liable to be misled by the measurement of the length of this organ, and conclude that he has to deal with an hypertrophied spleen.

Palpation is another highly important measure employed in diagnosing splenic diseases. Therefore the surgeon should remember that the spleen in its normal state is completely hidden by the natural curvature of the left side and the greater curvature of the stomach, where it is beyond the reach of palpation; but when the volume of the organ is considerably increased, it escapes from the curvature of the side and pushes up the stomach; then the hand may be depressed so deeply into the abdominal walls as to seize the inferior border and measure approximately the base of the spleen. The surgeon, however, in some cases, instead of discovering a hard, circumscribed tumor, may find a distinct sense of fluctuation, indicating the existence of an abscess, cyst, or swelling with a softening point (cancer, lympho-sarcoma, etc.). The same method of examination likewise enables the surgeon to determine the degree of mobility of the organ, the existence of adhesions, and certain kinds of friction.

Percussion is likewise highly serviceable in splenic examinations, since this organ is surrounded within the abdominal cavity, above, below, and on the inside, by organs giving out a resonant sound; but this normal resonance may be more or less obscured by corpulence. In percussing the spleen the patient should be placed in the right lateral decubitus, or, still better, in the standing position. It is highly important that the surgeon should keep in mind the fact that, while the lung, intestines, and stomach commonly yield on percussion a resonant sound, this resonance may become a source of error when the stomach or intestines by displacement cover the external surface of this organ. Furthermore, in the case of hepatization of the base of the left lung, the localization of the upper border of the spleen becomes very difficult, and frequently cannot be precisely determined. A similar source of annoyance may be found in the case of the inferior border when ascites exists, while in other cases the internal border may be obscured by the over-distention of the stomach with food or a tumor of the greater curvature. In other cases there may exist complete resonance over the whole splenic region, and this may happen when the spleen is either displaced or entirely covered with intestines or with a flatulent stomach. Finally, dulness of the spleen in some rare instances may be confounded with dulness of the kidneys, or even with dulness arising from a tumor in these organs.

Auscultation may sometimes be advantageously employed in marked hypertrophy of the spleen, which may compress the large vessels and thus produce a *bruit de souffle* resembling the sound sometimes heard in pregnancy.

Pain.—It has been asserted, but not yet proved, that the spleen in its healthy state is completely insensible to pain. But this statement is not here material, since it is admitted that the pathological changes in this organ are attended with pain. Pain may arise from any disease which produces a rapid dilatation of this organ, or it may result from a traumatism giving rise to a rupture. It will be found to exist under these circumstances in varying degrees, but is never constant or of very long duration. Pain is likewise frequently experienced in chronic diseases of this organ, but is also sometimes entirely absent. Pain in these chronic cases is commonly felt in the intercostal spaces and is frequently attributed to affections of the pleura or lung, while it is probably due to peritoneal complications.

Induration, fluctuation, or softening of the spleen may sometimes be determined by carefully percussing over the abdominal walls in the splenic region. Induration always exists in cases of chronic hyposplenitis. Fluctuation accompanies perisplenic abscesses, perisplenic peritonitic cysts, and likewise splenic cysts; and these morbid conditions may give rise to hydronephrosis, with a cyst in the left kidney or a pyelo-nephritis. Softening of the spleen frequently arises from congestion of this organ originating in malarial disease.

Enlargement of the Spleen, and its Complications.—These morbid conditions give rise to a great variety of symptoms, and therefore require a cursory examination in this connection.

Enlargement of the spleen attended with marked increase in its volume gives rise to important changes in its position. The inferior border is pushed inward and occupies the hypogastric region; consequently the vertical section, which has the greater diameter under the changed condition, assumes a transverse position.

The enlarged spleen possesses only slightly its normal bearings above, within, or without, but develops especially in the direction where it meets with the least resistance,—*i.e.*, inward and downward, coming in contact with the abdominal walls, pushing back the abdominal viscera, and even going so far as to displace the uterus and bladder. Furthermore, it occasionally pushes up the diaphragm, inconveniencing the left lung and heart, likewise the left kidney, stomach, and liver. The pushing up of the left lung, diaphragm, and stomach frequently gives rise to dyspnœa, while the displacement of the heart may produce a pleuritic effusion.

In other cases, when the intra-abdominal cavity is completely filled with a greatly-enlarged spleen, an ascitic effusion, or a gaseous collection, it pushes up the diaphragm and thus compresses the whole thoracic viscera. An effect somewhat similar to that arising from an enlarged spleen may be produced by a pleuritic attack with effusion. When the diaphragm is pushed down on the spleen, and this organ in turn is brought in contact with the left kidney, complications of this sort frequently lead to great difficulty in making a correct diagnosis, since the pressure of the spleen on the kidney produces symptoms closely resembling those which arise from tumors in the latter organ. Tumors of the left kidney cause, in fact, the same pressure on the spleen, and this gives rise to the same appearance of the abdominal walls. In this connection, however, a diminution in the quantity of urine possesses some value, since it may imply the destruction of the functions of the left kidney; nevertheless it should be remembered that the same result may be due to compression of this organ and its ureter by an enlarged spleen, thus mechanically producing the same result. Hæmaturia itself is a symptom of little value, since it may have its origin in leukæmic infarctions. The presence or absence of pus in the urine is, however, worthy of the surgeon's attention.

The quantity of urine, and the presence or absence of pus or blood in the urine, may be worth but little when taken singly, but will be found much more significant when studied connectively. In fact, if the quantity of urine voided in twenty-four hours is either normal or increased, it may be reasonably inferred that the kidney is not involved. If there has been a diminution in the quantity of urine voided, and no hæmaturia or purulent complication, it is highly probable that the true explanation of this morbid condition will be found in a mechanical pressure on the kidney and ureter. In other cases, where there is a diminution in the quantity of urine, with hæmaturia, but without a putrid odor or any other evidence of pus in this fluid, it may be assumed that it is a case of hæmaturia of leucocythæmic origin; while if there is a diminution, with hæmaturia and pus, it will generally be found that it is dependent on renal disease.

MORBID CONDITIONS REQUIRING OPERATIVE TREATMENT.

Perisplenic phlegmon, suppurative splenitis, gangrene, wounds, tumors, and *displacements of the spleen*, under certain circumstances require operative surgical treatment, and consequently demand here a brief description.

PERISPLENIC PHLEGMON.

E. Besnier has declared that perisplenic phlegmon is to the spleen what perinephritic phlegmon is to the kidney; but Maurice Jeannel has denied the correctness of this comparison, and attempted its refutation on anatomical grounds. He has called attention to the fact that the serous tunic enveloping the spleen is so intimately adherent to the fibrous capsule that it is impossible to detach it, except at the points where the ligaments are inserted, or on the posterior border, where there exists a little cellulo-adipose tissue; consequently, the only points where a perisplenic phlegmonous inflammation is possible about the spleen are at the insertion of the gastro-omental ligament into the hilus, at the insertion into the superior border of the phreno-splenic ligament, and on the posterior border.

It is claimed, in all cases of so-called perisplenic phlegmon, that the post-mortem examinations have shown that the parenchyma of the spleen has been found to be involved, although this does not prove that the primary disease does not first attack the coverings of the organ. This disease may arise as a complication of malarial or typhoid fevers, and, in fact, most frequently occurs in persons suffering from some marked cachexia. It may likewise arise in the course of pyæmia by the stopping up of a blood-vessel by a blood-clot, or by the extension of an inflammatory process from neighboring organs. It will be readily admitted that, while the etiology of this disease may possess considerable scientific interest, nevertheless the practical surgeon will feel much more deeply interested in those measures which are to be employed for the restoration of the patient.

This consideration of our subject brings before us all those questions which must be duly weighed in all operative procedures. The first question which should be settled in this connection is, Ought an operation to be performed? It is self-evident that no general answer can be given here, since every case must be carefully studied and the operative measures determined on its own merits. It is scarcely necessary to add, in this connection, that those having a pyæmic origin afford the surgeon very little, if, indeed, any, reasonable hope for the recovery of his patient. Let, therefore, the constitutional and local condition of every case be thoroughly understood before active measures are taken. The time when an operation should be performed is likewise very important, but on this point I shall attempt no discussion further than to remark that the safety of the patient will unquestionably be greatly increased if the peritoneum covering the pus-cavity has already become adherent to the abdominal walls to such an extent that no injury will arise from the entrance of the liberated pus into the peritoneal cavity. The preferable operation, even under these circumstances, consists in carefully cutting through the abdominal walls with the scalpel, under strict aseptic precautions, rather than attempting the evacuation of the pus-cavity by means of aspiration.

The perisplenic abscess, when no operative procedure is attempted, may remain encysted, but more frequently pushes inward along the peritoneum, when it is followed by an acute peritonitis and death; while under other circumstances the pus may push upward until it comes in contact with the stomach, when it produces ulceration and perforation of that organ; or, if it takes a somewhat different direction, the result may be a perforation of the diaphragm, followed by pleurisy, etc.

SUPPURATIVE SPLENITIS.

This disease has been studied from experimental, clinical, and pathological stand-points; but it is worthy of remark that the experiments of Gendrin consisted in the introduction of caustic substances into the centre of the parenchyma of the spleen in living animals, where it excited inflammation, which was followed by the formation of pus-cavities. These experiments, however, bear only on the etiology and pathology of the disease, without affording any assistance in our operative procedures.

Suppurative splenitis may be either of internal or of external origin, and may exist either as a primary or as a secondary lesion,—called secondary when the primary trouble has originated in a neighboring organ. The cause of the disease may be idiopathic, constitutional, or traumatic.

The disease may have its origin in an enfeebled constitutional condition resulting from malarial contamination or septic infection. The latter form, when it is purely pyæmic in its character, does not encourage any operative procedure, since the local manifestation is only a small portion of the constitutional disorder. It is thought that malaria may act as a predisposing or even as an exciting cause in the production of splenic abscess, by lowering the vital resistance or possibly by the production of an engorgement of the organ whereby the circulation of the blood is greatly impeded or even arrested in its vessels. Furthermore, when the distention of the organ has been very great and has been rapidly produced, laceration may have occurred with hemorrhagic infarctions, which play an important *rôle* in the development of the abscess. In the traumatic form of the disease the lacerations of the tissues of the organ and the resulting hemorrhagic infarctions are unquestionably closely allied to the inflammation and pus-formation which follow the injury.

The idiopathic form of this disease is unquestionably very rare, although its occurrence in connection with an enfeebled constitution has been admitted. The secondary or constitutional variety of this disease is certainly of more frequent occurrence than any or even all of the other forms. It may take its origin in an embolus, followed by the formation of a metastatic abscess, or in the propagation of a phlegmasia from a neighboring organ.

The symptoms of suppurative splenitis are not well marked, and consequently the diagnosis is always difficult and frequently of very questionable accuracy. Palpation reveals the fact that the tumefaction of the spleen is always moderate,—less than that which generally exists in chronic malarial hypertrophy,—while in some cases a careful examination indicates a point of fluctuation.

Pain is rare, exists only in cases of perisplenic peritonitis with adhesions, and is then felt in the left arm and shoulder. There may also be present a fever of the intermittent or remittent type. The extension of this disease to other organs gives rise to additional symptoms, which are characterized by the nature of the complication which has taken place: thus, when perforation of the stomach has occurred, pus may be vomited; when the kidney is involved, pus may be found in the urine; if the diaphragm is perforated, there will be developed symptoms of pleurisy or those of pleuro-pneumonia.

The prognosis is generally unfavorable, but must be made with reference to the case under consideration, while it should be remembered that when the visceral organs are involved it always adds to the gravity of the case.

Treatment.—The treatment is strictly surgical, in children as well as older patients, except in the pyæmic cases, where the author has recently employed large doses of the bichloride of mercury,—one grain a day to adult patients, which was continued several weeks,—with the most marked beneficial results and without the production of salivation or any other unfavorable symptom. Pathological investigations have shown that when an abscess is old it may become encysted by a proliferation from the fibrous tissue of the trabeculæ, the pus may be absorbed, the pockets contract, and finally there remains only a thin cicatrix in the centre of the splenic tissue. These observations encourage us to persevere in the treatment of splenic abscess of pyæmic origin, with the hope that by medication we may yet be able to arrest the disease and thus permit nature to effect a cure where the surgical art is entirely impotent. The surgical procedures required in these cases are either splenotomy or splenectomy,—both of which operations are fully described in another part of this article.

GANGRENE.

Gangrene of the spleen is an exceedingly rare disease if the word here employed is used in its ordinary sense; but the term is now often made use of to designate a condition of putridity which is of more frequent occurrence. It is with the spleen as with the brain, since neither is ever in contact with the air, except in the case of a wound, or when the latter organ by pathological changes has been brought in connection with the stomach or the intestines; therefore when it mortifies within the living body it does not undergo a true putrefactive change, but only macerates,—a process which is similar to what is observed in the fœtus under certain circumstances. Thus considered, gangrene of the spleen is closely allied to or perhaps identical with the softening of this organ (or splenomalacia) which is observed to follow an extremely pernicious attack of acute congestive disease. It is unquestionably this acute disease that primarily produces phlegmonous emboli in the spleen and the other resulting splenic changes. In regard to the etiology of gangrene of the spleen, the true explanation of its origin is always found in an acute congestion, followed by a high degree of inflammation attending the outbreak of certain pernicious diseases. The diagnosis is therefore made on the post-mortem table, and the treatment is *nil.*

RUPTURES.

Under this name are included solutions of continuity, involving either the capsule or the parenchyma, which are not produced by gunshot wounds or by cutting or pointed instruments. Rupture of the spleen may be traumatic, when produced by violence brought to bear on this organ, such as contusions over the splenic region, falls, etc. In other cases this lesion takes place in an organ already diseased, when the disease is to be regarded as the predisposing cause, while the immediate cause may be found in the ordinary movements of the patient, the movements of neighboring viscera, etc.

The traumatic rupture in a perfectly healthy spleen has been supposed to be of very infrequent occurrence, since this organ possesses a high degree of elasticity and is surrounded by organs which are very movable and likewise elastic. The author is, however, of the opinion that ruptures and lacerations of the spleen occur much more frequently as a result of injuries than has heretofore been supposed.

This opinion is based on a large number of post-mortem examinations made on dogs which had previously received traumatic injuries. This lesion occurred much more frequently in the liver than in any other abdominal organ, while the spleen took the second place in the order of frequency of occurrence, and the kidney stood in the third. It was likewise observed, in the cases to which I have just referred, that a rupture of the spleen was rarely found unless the liver was also ruptured.

Furthermore, these post-mortem examinations have fully convinced me that ruptures and lacerations, unless attended with grave hemorrhage, are very seldom diagnosed on the living subject, since spontaneous healing of these wounds promptly follows. It is undeniably true that the production of these lesions is strongly favored by certain pathological conditions, among which may be mentioned parenchymatous softening or perisplenic peritonitis with adhesions. In the latter cases it will be observed that, owing to the fixation of the spleen, the external violence will be enabled to act effectually, since the organ cannot slip away unharmed from the force of the blow. In cases of parenchymatous changes when the spleen has been rendered very friable, the ordinary movements of the patient, the movements of the neighboring viscera, or even those of the diaphragm, may produce lacerations. These ruptures may occur in any part of the spleen, may be single or multiple, superficial or deep, complete or incomplete, and in form and extent are necessarily variable.

Diagnosis.—No positive diagnosis can be made on the living subject in the milder forms of this lesion, and in the more serious ones the chief reliance must be placed on those symptoms indicating internal hemorrhage. Pain may be completely absent, or an entirely unimportant factor.

Treatment.—Hemorrhage is the chief danger in all cases of rupture, whether pathological or traumatic; consequently the treatment is mainly aimed at its control, especially in all the grave cases. The mild cases in which hemorrhage is not a dangerous complication may be expected to do well under rest in the supine position. The serious cases of traumatic rupture, which are always complicated with a dangerous loss of blood, should receive at the hands of the surgeon exactly the same general treatment which he would employ to control any similar hemorrhage within the abdominal cavity: *i.e.*, instead of relying, as heretofore, on the use of astringents, cold, etc., he should in certain cases promptly open the abdominal cavity and remove the spleen.

WOUNDS.

There is very little which it is necessary to say, at this point, in regard to wounds of the spleen made with cutting or pointed instruments. The diagnosis is easily made, but there is some difficulty experienced in their treatment, owing to the situation of the organ and its protection by the ribs. Gunshot wounds involving the spleen possess no peculiarities other than those common to the same class of injuries involving the organs of the abdominal cavity, except that of locality, which has already been mentioned in connection with incised wounds in this region. The principal danger in incised and likewise in gunshot wounds of the spleen arises from hemorrhage, which may be either primary or secondary. The danger from secondary hemorrhage and inflammatory complications is almost entirely avoided by a strict adherence to an aseptic toilet and other aseptic measures, as has been fully demonstrated by the recent practice of abdominal surgery. The blood extravasated beneath the capsule may either be absorbed or result in the formation of a splenic cyst.

Diagnosis.—The diagnosis in former times was based essentially on the locality of the wound and indications of hemorrhage; but the time has now been reached when the surgeon should unhesitatingly, under certain circumstances, enlarge the wound in the abdominal walls and thus determine the extent and character of the injury.

Treatment.—The old treatment employed in the management of these cases, in which the spleen was found protruding through the wound in the walls of the abdomen and was there left to unaided nature or merely assisted by the application of a ligature to the protruding viscus, can no longer be recommended. The proper course to be pursued under these circumstances must depend on the extent and character of the lesion. Should it be found that the spleen is irrecoverably injured, splenectomy should be immediately performed; but when this organ is still in a viable condition it should be thoroughly cleaned and returned to the abdominal cavity, even though it may be necessary to enlarge the opening in the abdominal walls.

Care should be taken to control all hemorrhage and to cleanse perfectly the abdominal cavity. The drainage-tube should then be introduced, the external wound closed with metallic sutures, and a perfectly aseptic dressing applied, and above this a few bandages, when the patient should be directed to lie on the wounded side. Should inflammatory or other trouble arise, it should be treated according to the general rules of surgery.

DISPLACEMENTS.

The spleen is fixed in the abdominal cavity by two ligaments, which have been fully described in that portion of this article which is devoted to the anatomy of this organ. These ligaments, by stretching or lengthening, occasionally allow this organ to occupy anomalous positions, whether it be healthy or diseased. Thus we find the spleen sometimes in the hypogastric or the umbilical region, the iliac fossa, or the pelvis. The spleen thus displaced forms new attachments to other organs with which it may come in contact, but it is nevertheless recognizable in its new situation by its form and by the dulness which it gives to a region which should otherwise be resonant. The old site of the spleen is filled with intestines, which produce a change that may be recognized by palpation and percussion. The symptoms are sometimes those of compression or obstruction of the abdominal viscera. These displacements are invariably attended with much pain, caused by irritation or other injurious impressions made on the splenic nerves. This condition is rarely seen in young children, but has been observed by the author in adolescent and adult females only; and therefore we are brought to the consideration of the question, Why should the female alone thus suffer?

In my attempt to solve this question I shall call the attention of the reader to those factors only which it seems probable may have a more or less important bearing on this topic. The evil practice of tight lacing, by which this portion of the female body is so greatly constricted, is unquestionably the chief cause in the production of this malposition, while it is thought that the want of the proper development of the muscles of the female abdomen, which must necessarily result from neglect to take proper exercise, as well as the variations in the degree of pressure exercised by the visceral organs, pregnancy, etc., upon the muscles of this portion of the body, may likewise have some influence on its causation.

Treatment.—The use of medicine here is intentionally restricted to those cases in which the displacement is complicated with a malarial enlargement or some other diseased condition of the organ. The surgical treatment consists in the restoration and retention of the organ in its normal position; the latter is accomplished, as far as possible, by the employment of bandages and the position of the patient's body. The majority of cases may be relieved by these measures, but the most aggravated may justify the performance of splenectomy.

TUMORS.

Tumors of the spleen may be either liquid or solid. The *liquid tumors* of the spleen are cystic in their character, and generally situated in the centre of the parenchymatous tissue, although they are occasionally found on the surface of the organ. These cysts are either serous or hydatid. The serous cysts may be either solitary or multiple, similar to those which have been observed in the neck of the uterus.

The hydatid cysts are analogous to those found in the liver, and possess clinically all their peculiarities. They are commonly developed just beneath the capsule of the spleen, and buried on the surface of the organ within the parenchymatous tissue.

The diagnosis is generally difficult, and frequently impossible.

Their treatment does not require any special consideration.

The *solid tumors* occupy either the capsule or the parenchyma of the spleen. The tumors of the capsule which have been examined have been either myomatous or fibro-cartilaginous. The lipomatous tumors are developed in the cellulo-adipose tissue in the neighborhood of the hilus. The tumors of the parenchyma are the lymphadenoma, splenadenoma, and spleno-lymphoma, which belong especially to the Malpighian corpuscles. They are coincident with the general glandular hypertrophy which Cornil and Ranvier describe thus: "The Malpighian corpuscles, which represent the follicles of the lymphatic ganglions, are immoderately hypertrophied. They occasionally reach the size of a hazel-nut, or even of a walnut. The reticulated tissue presents almost everywhere the thickness of its trabeculæ, with hypertrophy of its voluminous nodes. The capillary blood-vessels are fully distended with white globules in cases of leucocythæmia, cancer, etc."

CANCER.

Primary cancer is exceedingly rare in this organ, while a secondary development is of somewhat more frequent occurrence. The primary cancer in these cases is generally encephaloid, while the secondary commonly corresponds to the analogous tumor in the liver or stomach. The most characteristic symptom of this disease is a very severe pain in the splenic region when the disease is known to exist in some other organ of the body. However, there are cases of cancerous disease of this organ in which the correct diagnosis is revealed only by the post-mortem examination.

TUBERCLE.

Tubercular deposits in the spleen are frequently met with as a complication of tubercular disease in other organs of the body.

OPERATIVE TREATMENT.

The operative treatment required of the surgeon on this organ is essentially limited to aspiration, exploratory laparotomy, splenotomy, and splenectomy.

ASPIRATION AND EXPLORATORY LAPAROTOMY.

Aspiration should never be employed in the treatment of any disease of the spleen until the surgeon has satisfied himself that there is an abscess-cavity within this organ and that the capsule of the same is firmly adherent to the abdominal walls. If this rule be strictly obeyed, there will be found very little use for the aspirator, and I am fully convinced that in all cases where it is desirable to obtain additional information for diagnostic purposes an exploratory laparotomy should be performed, as the safer and more useful operation. The dangers inseparable from the use of the aspirator in abdominal surgery, as well as the comparative advantages of an exploratory laparotomy, have been so recently and so fully discussed in medical literature that I may be excused from enlarging upon them.

SPLENOTOMY.

The term splenotomy (preferably laparo-splenotomy) was until recently extensively employed to indicate the extirpation of the spleen, but it is now generally restricted in its application, and is used to designate those cases only in which an incision is made into this organ for the purpose of liberating pus, serum, etc. This operation unquestionably may be wisely resorted to in the treatment of superficial abscesses and cysts when the bulk of the organ is still in a comparatively healthy condition ; but neither deep nor extensive incisions should be employed, since its anatomical structures are such as to present insurmountable obstacles to the control of hemorrhage.

The incision having been made through the abdominal walls at the same place as that recommended for splenectomy, and the diseased organ thus brought under the surgeon's eye, the bulgings of the capsule at a single point or at many points will generally fully satisfy the surgeon whether splenotomy or splenectomy is the preferable operation, since it is evident that the former operation should give place to the latter in all cases of general cystic degeneration of the organ. Splenotomy therefore should be preferred in certain cases of simple or hydatid cysts, abscesses, etc., and during its performance the same care should be exercised to prevent septic infection as is employed by the most painstaking operators in the performance of other operations within this cavity. While the surgeon is engaged in liberating pus or other fluids contained within the diseased spleen, he should exercise all possible care to keep it from coming in contact with the peritoneum, the visceral organs, or the cut surfaces of the abdominal walls.

Having emptied the pus-cavity, it should be carefully examined, afterwards rendered aseptic with all the adjacent parts, and the entire length of the incision in the spleen carefully stitched to the margins of the wound of the abdominal walls. The wound is then prepared for the introduction of a suitable drainage-tube, which is promptly followed by the closure of the wound in the abdominal walls and the application of an aseptic dressing. The after-treatment consists in removing, as frequently as may be necessary, the fluid accumulated in the tube, and likewise preserving asepsis in the wound and surrounding parts.

SPLENECTOMY.

The complete and accurate history of splenectomy (or, more correctly speaking, laparo-splenectomy) would undoubtedly be very interesting and likewise instructive, but our limited space does not enable us to enter fully into these details. Dr. S. W. Gross, of Philadelphia, who recently made a very careful historical and statistical study of this operation, expresses the opinion[1] "that a diseased spleen was first removed at Rostock, in Germany, by Dr. Quittenbaum, only fifty-one years ago,—*i.e.*, October 5, 1836;" and he finally concludes that, "as the entire spleen has been removed because of disease or displacement, or both in combination, some sixty-two times, and the proportion of cures is decidedly on the increase in latter years, we can readily afford to confine the record to an enumeration of the cases known to have been of a legitimate character. The leukæmic spleen is the most hazardous to remove, and, if the leucocythæmia is very decided, the operation is always fatal. A simple hypertrophic spleen, recent or congenital, is less dangerous to exsect, and the displaced or 'wandering' spleen is least. But in all the operations the risk from hemorrhage and shock is very great, and patients are liable to die in collapse within a few hours. In traumatic cases there is much less risk in the removal of the organ, and the records of the past are decidedly encouraging to the operator."

The following statistics may be employed (at least approximately) in fixing a basis on which the surgeon may select or reject a case for splenectomy, since this operation has been performed with the results shown in the following table:

	Cases.	Recoveries.	Deaths.	Causes of Death.
Cysts	5	4	1	Not known.
Sarcoma	1	1	..	
Wandering spleen	13	11	2	Collapse. Twisting stomach.

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[1] Medical News, January 21, 1888, p. 74 *et seq.*

	Cases.	Recoveries.	Deaths.	Causes of Death.
Simple hypertrophy (so called)	14	1	13	Hemorrhage, 8. Peritonitis, 1. Septicæmia, 1. Cirrhosis, 1. Shock, 2.
Malarial hypertrophy	3	..	3	Septic peritonitis, 1. Shock, 1. Parenchymatous nephritis. 1.
Leucocythæmia	20	1	19	Hemorrhage, 14. Shock, 3. Septicæmia, 1. Exhaustion, 1.

It is further shown that operative procedures in cases of traumatisms have been attended with marked success. Nussbaum reports twenty-six operations for traumatic injuries, with sixteen recoveries. Gilson gives us eighteen similar operations, with eighteen recoveries. Ashhurst reports twenty-one operations for injury or prolapse, where all recovered. It cannot be denied, even if some of these statistics are not strictly accurate, that they furnish us with valuable information, affirmative as well as negative, in the selection of suitable cases for surgical interference. We therefore on this basis conclude that operative surgical interference may be demanded in the following splenic conditions: (1) traumatic injuries, (2) displaced or wandering spleen, (3) malignant disease (early stage), (4) serous or hydatid cysts, (5) abscesses, etc.; but that it is contra-indicated in all cases of leucocythæmia or malarial enlargement.

Statistics further show that the principal sources of danger in all these operations are hemorrhage and shock, while the former is very closely connected with the pathological changes, both local and constitutional, which accompany leucocythæmic and malarial enlargement of the spleen. The extirpation of the spleen is never free from danger, and therefore an operator should give to this subject the most careful consideration. It is a fact well known to every surgeon that the laity are always ready to express an opinion on the merits of our professional work. When a foolhardy attempt succeeds, they are ready to worship the operator; but, should a well-planned and well-executed operation fail, the surgeon will frequently be denounced as a murderer.

Operation.—The preliminary preparation for the performance of this operation should be made with the same care as is practised by the most painstaking aseptic surgeons in their operations within this cavity. In fact, nothing should be omitted in the preliminary preparation or during the performance of the operation which could in any degree increase the chances of the patient: especially ought the surgeon to exercise the highest degree of skill in the control of hemorrhage, lessening of shock, and preservation of asepsis, since this is the tripod on which he must base his operation if he expects success.

The point at which the external incision ought to be made should in a measure depend on the circumstances attending the operation. In the case of cysts, cancer, etc., and, in fact, in all cases when there is neither displacement nor wound of this organ, nor a solution of continuity involving the abdominal walls, the incision should commence one inch below the costal cartilage, and be carried downward to the external border of the left rectus muscle from four to seven inches. The incision should be carefully carried through the abdominal walls, and the precaution taken to ligate every bleeding vessel before opening the peritoneal cavity.

The peritoneum covering the spleen is then divided on the grooved sound, after which the superabundant fluid of the peritoneal cavity is allowed to drain off. If the diseased spleen is adherent, it will require the exercise of *extreme care* to break up these adhesions without causing a laceration of the organ, making traction on the pedicle, or doing injury to the splenic plexus of nerves,—either of which accidents might cause the death of the patient. The accidental protrusion of the intestines through the wound in the abdominal walls is liable more or less to embarrass the surgeon, although in some cases the spleen is pushed forward, which places it in the most favorable position for the operative procedure. If it should happen that the surgeon while cautiously separating the adhesions should have caused even a moderate hemorrhage, he should immediately arrest it, in order that he may have at all times a clear view of the field of the operation.

The lower portion of the tumor should be first freed, and, while this is going on, aseptic sponges having a string attached to each may be packed within the cavity, while the strings are to be carefully kept outside of the wound. These sponges are so placed as to keep back the intestines, to steady the spleen, and likewise to prevent traction from being made on the pedicle, while the strings enable the surgeon to determine when all the sponges have been withdrawn from the cavity.

The most difficult and likewise the most important part of the operation consists in the effectual ligation of all vessels of the pedicle. These vessels all converge and enter the hilus of the spleen, and the effort of the surgeon should be to reach them without doing harm, and, if possible, ligate each vessel separately, placing the ligature so firmly that it will not slip from its position, and to divide no vessel until all have been secured. It is as important to ligate the veins as to ligate the arteries, while the nerves are carefully excluded from the vessels. In some instances it may be found advantageous to employ hemorrhage-forceps, clamps, etc., in the performance of this operation. These forceps may be applied to the vessels in the pedicle in such a manner as to leave a sufficient space between them for the application of the ligature, but it would seem more proper to apply the ligatures before the vessels are divided. It may further be added that in all cases where either the clamps or the forceps are employed their use is only temporary, and that when all the vessels have been perfectly secured the pedicle is returned to the cavity.

The gastro-splenic and the diaphragmatic ligaments are then cautiously divided, but not until their blood-vessels have been properly secured. The cavity should then be thoroughly cleansed with hot water, while the surgeon very carefully avoids any unnecessary handling of the pedicle, lest he might thus excite hemorrhage by the slipping of a ligature or by other avoidable complication. He should ordinarily wait a few minutes after the removal of the spleen, so that if any oozing point is discovered it may be properly treated. The drainage-tube should then be introduced, and the wound properly sutured, including the peritoneum. The dressing should be perfectly aseptic, and the patient should be placed in such a position as to afford the greatest degree of immobility to the injured parts.

ADENITIS (SCROFULOUS GLANDS).

By SAMUEL ASHHURST, M.D.

My theme is non-malignant enlargements of the lymphatic glands as occurring in children, which, though observed in widely different localities, are either simply congestive or inflammatory. Among the former must be included all those so-called sympathetic enlargements of glands so common and so often evanescent. Among the latter will be found all those modifications of inflammatory action peculiar to lymphatic glands which are regarded as scrofulous in their essence.

As preliminary, a few words concerning the anatomy, physiology, and pathology of the lymphatic system will not be out of place. Beginning apparently by open mouths in the interspaces of the areolar tissue, the lymphatics appear first as condensations of that tissue serving as channels to convey into the circulation whatever foreign substance or effete material has found entrance to any areolar interspace. Whether in solution or in merely minute mechanical division, the substance is seized upon and carried towards the circulatory system, with the important difference that, while bland or soluble matter is carried on without hinderance, irritating or insoluble substances appear to suffer detention in the nearest glands and to act in a greater or less degree as direct irritants to the tissues entering into the substance of the gland itself.

The lymphatic gland seems to be nothing more than an expansion of a lymph-channel, and is often unrecognizable in a state of health, though filled, as are the lymph-channels, with lymph-corpuscles, and surrounded by a capsule apparently composed of the fibrous tissue condensed and thickened as its contents expand and enlarge. Where these lymph-corpuscles come from is not positively determined; but they must originate either within the lymphatics themselves by cell-proliferation, come from a starting-point of inflammation, or be white corpuscles which have leaked out of the capillary vessels. Under the influence of irritation there is a vast increase in the number of these corpuscles, adding to the size of the gland, until what was frequently at first so minute as to defy casual detection is now evident as a globular tumor well marked and of perfectly defined limits. Very rarely will such a gland be found alone. Almost always, whatever may be the impression arrived at from external examination, dissection will reveal the existence of others; while very generally they exist in great numbers, more or less matted together, and forming the well-known and familiar masses so slow to disappear and so apt to cause marked deformity of a permanent character.

The afferent lymphatic vessels enter the gland between the layers of the capsule, and accordingly the first part to be subjected to injurious influence from without is the capsular plexus of the gland proper. It might therefore be inferred that inflammatory changes would first manifest themselves in the superficial portions of the gland. But experience proves that this is not the case, the progress of the affection being originally most evident in the deeper portions of the gland, beginning first in the medulla and extending thence to the cortical portions of the gland proper, and never invading its capsule. These changes manifest themselves as spots of varying shape and size, which do not take as deep a stain from hæmatoxylin as other parts of the gland, and exhibit to the examiner many diverse cellular forms, from true lymph-corpuscles to large, conspicuous cells containing nuclei, which are transparent, and show most distinctly an intranuclear plexus. Many gradations between these two forms show that the large cells are lineally connected with the true lymph-corpuscles. All these cells give evidence that active division is going on, while the endothelial cells appear to be unaltered. The capillaries in the part are very numerous, and exhibit an adenoid sheath, which, though described as histological, is best seen when some inflammation is present. The large cells are a very prominent feature in scrofulous inflammation, and their appearance and multiplication by segmentation are essential factors in the diseased process. As that process advances, these cells undergo no further development; they never become the giant cells observed later, but speedily degenerate and disappear.

Next changes are observed in the lymph-sinuses near the medulla. The endothelial cells lining them increase in number and size. The reticular arrangement within the lumen of the lymph-sinuses is hypertrophied as to the size of its fibres, and their number is increased. Within the meshes of this reticular structure are scattered leucocytes, some normal, some having undergone the changes already spoken of, with some of the large cells described by Rindfleisch. At the same time that these alterations are being made, the gland-tissue proper is becoming crowded more and more with the same cell-elements, and is becoming opaque from the deposit, beginning near the lymph-sinuses, of coagulable lymph. As the opaque spots thus formed increase in size, anatomical details become indistinct, increasing in size by peripheral extension, and are always rounded in outline. These spots increase in number and gradually impinge upon one another until several coalesce to form one of the lobulated and more or less circular patches so readily discernible in these cases on the most superficial examination. Within these patches the cell-elements undergo fatty degeneration, the large cells going first, while the small endothelial cells may be detected until the general degenerative change is comparatively far advanced.

The above description applies to glands in which the change is somewhat active. Where the process is more indolent, the general changes go on more slowly, and there is a distinct tendency to an increase of fibrillation. The cells are more varied in size, and, from the increased amount of fibrillar tissue, appear to be less numerous. The same increase of fibrillar tissue also makes the opaque patch more homogeneous in appearance.

Within these opaque patches, the *îlots strumeux* of Cornil, the well-known giant cells make their appearance. They are not the forerunners of inflammatory change, not appearing until the comparatively advanced stage that has been described. The character and *raison d'être* of these giant cells have been much discussed. As has been intimated, they cannot be looked upon as active agents in producing inflammatory changes, being rather the products of changes already considerably advanced. The suggestion of Treves is that they are merely lymph-coagula involving more or less numerous cell-elements in their coagulation. This suggestion seems reasonable from the fact that the material of which they are composed appears to be identical with other lymph-coagula. They are often found occupying the lymph-sinuses, and would very probably be found there much oftener were it not for the fact that the anatomical details are so generally rendered indistinct by the rapid progress of the degenerative changes. They very closely if not precisely resemble the giant cells which are sometimes found in chronic inflammation of the connective tissue, and which are admitted to occupy the lumen of lymphatic vessels. There they make their appearance at the same time that much coagulated lymph pervades the gland. There are other reasons militating against the theory which regards these "giant cells" as protoplasmic masses, but they would be out of place in an article necessarily so limited as this.

Caseation begins in these opaque patches, it being essentially a process of fatty degeneration accompanied with desiccation of the part. It begins in the centre and proceeds outward, and, where it is at all complete, all that is to be seen is fatty matter and granular débris, with the occasional remainder of what has been a cell. Sometimes the caseous matter gradually dries up into an inert mass; more generally it liquefies, and, by irritating the adjacent parts, induces suppuration, and abscesses follow.

To the naked eye glands which have advanced so far present a pale flesh color upon section; the caseous masses can readily be felt when they are at all advanced, and project more or less from the surface of a section which has gone through them. They can sometimes be detected where the gland is held up to the light. Especially is this true where the cheesy nodule is proportionately large, and the glandular tissue has become paler, as it does with the progress of the disorder. The amount of caseation is independent of the size of the gland. A small gland may be almost entirely transformed, while, on the other hand, in one much enlarged the caseous change may be but slight. Glands in which the caseous change is not at all proportioned to their increased size are those which are sometimes spoken of as hypertrophied.

In many cases the morbid action is much slower and the tendency to the formation of fibrous tissue is marked. This fibrous tissue tends to form in circular masses,—so-called tubercles. There is an appearance of much solidity, and cell-forms are either very scantily present or much withered. The interspaces (tubercles) contain some unaltered lymph-corpuscles, and homogeneous material much resembling coagulated lymph. Sometimes giant cells can be seen in the centres of the more or less circular intrafibrillar spaces. Treves argues that in these cases, also, the giant cells are merely lymph-coagula, that the appearance of dimly-seen fibres, as they undergo softening and change, is caused by the lessened obstruction to the view, and that the fibres are rendered visible as the lymph-masses dissolve, being really continuous with the fibrous reticulum of the gland-tissue.

To the naked eye the glands last described appear less vascular, the section is more opaque, when suppuration occurs it is diffused, and an abundance of fibrous tissue is always discernible; especially is this last change perceptible in the capsule of the gland.

It will be noticed that two classes of glands have been described,—the scrofulous and the so-called tubercular of Cornil and some other pathologists. Treves and some other careful observers regard the differences as of grade rather than of kind.

The clinical differences between the two classes are marked. In the class first described the progress of the disease is more rapid and may be accompanied by more evidences of inflammation. The enlargement of the glands is greater, they tend to become matted together, caseation occurs at a comparatively early day, and suppuration is a pretty constant attendant. In the class last described the progress of the disease is more indolent; many glands may be enlarged, although they are not generally large individually. The glands, owing to the absence of inflammation, do not mat together, but remain movable. Caseation makes its appearance at a late day, and there is little tendency to suppuration.

In some cases the disease may spread from gland to gland by the mere continuance of the peripheral irritation conveyed by other lymph-channels, or the glands may have been simultaneously infected, though the manifestations of disease may not be equally rapid in different glands. In other cases the disease may first show itself by enlargement of a gland remote from the point of irritation, and the intervening glands be affected later on. These phenomena may be explained on the theory of a damming up of the lymph-current, and the later implication of the nearer glands by a backward pressure. In other cases the order of the progress is such as is easily accounted for by the continuity of parts. Or the irritating cause may find its way from the gland-tissue into the encompassing capsular plexus of lymphatics, and thence into afferent vessels, which take upon themselves the same development as other lymphatics. Again, the numerous glands, made evident by disease, which are yet entirely unknown to the student of normal anatomy, should be remembered, as contributing to strengthen the theory which accounts for the spread of lymphatic disease by continuity of tissue.

Etiology.—The causes of glandular disease are constitutional and local. The former cause has been long recognized, if not unduly magnified, by the profession. The latter has come into prominence of late years with the better pathological knowledge, of which an outline has just been given, and has in turn been elevated in importance, even to the well-nigh total exclusion of the former. The truth probably lies midway. That there is in many persons a delicacy of constitution, inherited or acquired, which predisposes to glandular disease as well as to other forms of disease, hardly seems to admit of doubt. This constitutional delicacy or weakness has been long recognized as the underlying cause of multiform manifestations known as struma or scrofula. Its existence would seem to be essential to the development of prolonged glandular disease, or of that form of it which proceeds to caseous degeneration of tissue. Persons in ordinary health may have temporary engorgement of a gland, the result of peripheral irritation, and that engorgement may go on to inflammation and suppuration, either in the gland or in the tissues around it; but that slowly-advancing structural change of which the histological history has been given is not found in persons who do not possess the constitutional predisposition referred to. Indeed, daily experience goes to show that even temporary and acute glandular engorgement is in large measure dependent upon the condition of the general health. Every surgeon knows how different are the effects of dissection-wounds at different times, and that any even temporary condition of impaired health adds much to their gravity and importance. When, therefore, there exists a permanent condition of more or less imperfect health, with the resulting impaired ability to resist tissue-change, it is reasonable to expect pathological alterations.

But mere depression of vital power would not seem to be sufficient to produce the conditions known as scrofula, else we should find every cachexia leading to struma. That there is in addition a peculiar element, *sui generis*, essential to that condition of things known as scrofula, seems to be an unavoidable inference. Such an inference is in accord with long-observed facts, and is analogous to the order of things as seen in many other affections. The close connection between that congener of scrofula, true tubercle, and the microscopical bacillus, apparently rests upon too secure a basis to be entirely denied, and there seems no reason to question the probable existence of some other germ which may represent the essence of struma.

On the other hand, the tendency of modern investigators is very positively towards the recognition of local causes as the sole foundation upon which the structure of scrofula rests. It has become pretty well established that glandular engorgement is hardly ever primary, but almost if not quite always dependent upon a peripheral lesion of the part from which the lymphatics going to a particular gland are derived. The glandular disease is therefore a secondary one.

Some writers regard this as a fixed rule without exceptions; others, while admitting its general correctness, are of the opinion that exceptional cases exist, and that glandular disease may therefore come into being without the pre-existence of a primary peripheral lesion. While those thus arguing lay themselves open to the charge of basing a theory upon a negation and upon defective observation, there are undoubtedly cases which favor the view, and which can be connected with an initial lesion only by greatly stretching the doctrine which maintains the invariability of such a connection. At least this is the case at present. With greater knowledge the link in the theoretical chain, which is now apparently wanting, may be found, and the reality of the connection proved. These so-called exceptional cases present a quite uniform picture and closely resemble one another. The hereditary history is marked, as is especially true of the tendency to phthisis. The gland-disease, while insidious and slow in action, is very wide-spread. Yet while the glands of many localities are involved, and large aggregations of glands are formed, the glands individually are not generally of large size, and when examined they are found to present invariably those conditions which have been described by Cornil and others as tuberculous. But these cases, it must not be forgotten, are very rare, and should not detract from the importance of the rule which places glandular disease in the category of secondary affections.

The bearing of this rule upon the question of treatment will be readily seen to be most important, and it should receive the most careful and minute attention. For that is unquestionably the most satisfactory treatment of glandular disease which is preventive, and which by attention to often-neglected peripheral lesions hinders engorgement of the glands.

The primary lesions which may induce glandular enlargement are numerous, and belong to many localities. The bronchial glands often undergo enlargement, owing to the presence of bronchitis, especially when it has been associated with measles, while the mesenteric and abdominal masses may take on enlargement as the result of catarrhal inflammation or ulceration of any part of the mucous membrane of the alimentary canal. Indeed, few scrofulous children are free from enlargement of the abdominal glands, which is readily accounted for by the great tendency of such subjects to suffer from gastro-intestinal disturbances.

Enlargement of the cervical glands may be dependent upon a great variety of causes. Among these may be mentioned eruptions and ulcers of the skin of the face and scalp, stomatitis, inflammatory affections of the fauces and pharynx, coryza, diseases of the ear, and imperfect dentition. Naso-pharyngeal catarrh is extremely apt to induce glandular disease, which in great measure explains the frequency with which this affection follows measles and scarlatina. Injuries and the inflammatory changes which follow them may readily induce enlargement of the neighboring glands.

For a long time it was thought that exposure to cold was a fruitful source of adenopathy, but the drift of modern investigation tends to attach less importance to it as a producing cause.

Treves gives the following table of the comparative locations of gland-disease:

Neck alone	131	Axilla alone	4
Neck and axilla	12	Neck and groin	1
Groin alone	6	Neck, groin, and axilla	1

The reason for this very large preponderance of cases where the neck is the part involved would seem to lie in the proximity of the mucous membrane lining the nose, the mouth, the pharynx, and the throat, and the prevalence in those parts of a large amount of submucous, adenoid tissue. The neighborhood of the tonsils, the largest masses of adenoid tissue in the body, by their frequently diseased conditions, would alone seem to be a fruitful source for supplying to the cervical glands that irritation which is necessary to excite them to take on enlargement and strumous degeneration. Experience shows, at least, that enlargement of the cervical glands will much more speedily and much more readily follow disease of the tonsils or of parts where there is much adenoid tissue, than it will the presence of comparatively severe disease in parts not so supplied. The marked difference in the liability of the glands to become enlarged in accordance with the fact of the initial lesion being in the immediate neighborhood of adenoid tissue can be readily observed by comparing the infrequency with which it occurs when the disease is upon some external surface, as the cheek, with the frequency of the complication when the primary trouble is in the mucous membrane of the mouth, nose, or pharynx. In seeking to find out the exciting causes of glandular disease, it must not, however, be forgotten that not only must the constitutional predisposition exist, but also that the inexplicable personal peculiarity known as idiosyncrasy will play an important part in determining the development of the affection.

Symptoms.—While there exist many varieties both in the appearance and progress of cases of scrofulous glands, there are general characteristics that are present with more or less regularity in most instances. The disease begins insidiously and without pain, so that its presence is most generally made known by the discovery of a lump, often of considerable size, but without heat or other inflammatory symptoms. The more marked the strumous diathesis the more pronounced is this chronic and indolent feature, and with rare exceptions, when there are acute symptoms, the scrofulous character of the disturbance may be eliminated from the case. Neither should it be forgotten that in some children without other marked strumous characteristics there will sometimes be found a chain of slightly-enlarged cervical glands which are prone to become tender under any temporary and local irritation, but which tendency disappears with the disappearance of the temporary cause. Sometimes this tendency disappears altogether at puberty; but the writer has met with instances where it has continued, with diminishing force, into adult life.

Sometimes but one gland apparently is affected, while at other times many glands may be simultaneously involved. Generally the glands are at first tumors, firm, elastic, and painless, over which the skin moves freely, and this condition may continue unchanged for a long period. There is a tendency for neighboring glands to enlarge and the whole number to become matted together. No further change may be perceptible for a long while, but after a greater or less length of time signs of inflammatory action appear. But even this is of a low grade, and the formation of pus is unaccompanied by any of those symptoms which ordinarily attend suppuration. The formation of pus, also, may not take place for many years, the glands undergoing several changes in the mean time, inflammatory symptoms appearing and then disappearing at intervals. Ultimately, however, suppuration takes place, but still the grade of inflammation is low, with thinning of the skin, but not much heat, and, although the presence of pus is very perceptible to the touch, there is little tendency for the abscess to point and for its contents to escape unassisted. But in very many cases, and especially where proper treatment has been pursued, the glands undergo resolution. This may take place at any time, and its occurrence is not rendered improbable by the fact that there have been one or more exacerbations of the disease threatening suppuration. The condition of the general health has much to do with determining whether suppuration takes place or not, and this termination is a less likely result in the case of children. When suppuration does occur, it may be either in the gland itself or in the areolar tissue around the gland, and, though the processes are generally found in practice to be combined, they are sometimes separated and present quite different features. When the suppuration is confined to the gland proper, the abscess may be made out as a limited, fluctuating point, surrounded by more or less condensed fibrous tissue, while the pus is thin, ill formed, and flaky, containing cheesy masses, and, when the interior can be seen, it is found to be ragged, as would be looked for in a disorganized gland.

When the suppuration is around the gland, rather than in it, the symptoms are more those of an ordinary abscess with laudable pus. When the opening is enlarged, the diseased gland can generally be detected at the base of, or in the wall of, the abscess, and most commonly the abscess will not heal while the gland remains. When a part of the diseased and disorganized gland does not actually prevent healing, it would appear very often to be the focus and starting-point of a future residual abscess.

The cicatrices following scrofulous abscesses are generally irregular, elevated, and conspicuous, often resembling those which follow burns. The color is dark, and there is generally for a long time much tenderness, with a tendency for the scar to ulcerate upon any deterioration of the general health.

Sometimes the enlargement of the glands is so great as to interfere with neighboring and vital parts by pressure. Instances of this kind will be found in many surgical works. They are, however, exceptional, and the writer has nothing to add to the subject as the result of personal observation.

Diagnosis.—There is little difficulty in deciding the character of a glandular tumor. Its occurrence in childhood in the majority of cases, its site, most often cervical, its persistence, its indolence, its tendency to the formation of caseous pus, and the presence very often of other scrofulous affections, are ample grounds upon which to form a very clear diagnosis.

Treatment.—This is both constitutional and local. In the majority of cases the general treatment is the most important, as if it is adopted early and carried out persistently there will in many instances be no occasion for any further treatment. Experience shows that fresh air, sunlight, good ventilation and drainage,—in other words, good sanitation,—constitute the most efficient aids to prevent the development of the scrofulous diathesis, and the best curative treatment where it has been developed. Especially is a residence at the sea-shore beneficial. This fact has long been recognized by the profession, and has found practical expression in the establishment of infirmaries at the points nearest on the coast from large cities in Europe and this country. The Children's Hospital in Philadelphia has for a long time availed itself of the advantages offered by the Sea-Side Home at Atlantic City, and every succeeding season bears testimony to the good effects derived by some of the severer cases from a longer or shorter residence in sea air. But it is in cases where the disease is incipient that the greatest benefit can be derived, and, where the circumstances of the child admit of it, a long-continued residence upon the coast will be found most beneficial. In selecting a proper location, attention to the general sanitary conditions should not be neglected, and, except in midsummer, the more southerly resorts will be found generally to offer the greatest number of advantages.

While the testimony of medical writers at large bears evidence to the virtues of sea air, there is equally positive proof that, where a residence upon the sea-coast is not obtainable, fresh country air is most advantageous, and this fact should be primarily taken advantage of in the treatment of these cases when occurring in close or crowded cities, and where the circumstances of the patient permit of its adoption.

The medicinal treatment, after passing through many changes, has of late years come down pretty much to the administration of nutrients and tonics. Of these the most beneficial are cod-liver oil, preparations of malt, iron, and one of the cinchona alkaloids. Experience has not proved that iodine possesses the efficacy once claimed for it. Yet, combined with cod-liver oil, there is no one remedy more properly relied upon than the syrup of the iodide of iron. The addition of a simple tonic is important as an aid to the digestion, and careful attention to this point is of much importance. Minute doses of calomel and bicarbonate of sodium occasionally are useful in keeping the bowels in proper condition, and the combination of the alkali with one of the bitter tonics is a most useful aid in improving the digestion and enabling it to bear the cod-liver oil.

While the general condition of the patient is improved by these means, they exert little if any immediate influence upon the enlarged glands, and it may require years of patient waiting ere the latter diminish in size or tend to disappear. Yet, so long as they do not enlarge or inflame, the conservative surgeon will be satisfied with doing little more. At least that was the disposition formerly. Of late, however, from the opinion that these diseased glands are in reality foci of infection, there has been more inclination to interfere with them.

This brings us to the consideration of the *local treatment* of enlarged scrofulous glands. Tincture of iodine painted upon the overlying skin does not accomplish as much as was at one time supposed, and, while sometimes beneficial as a counter-irritant, in diminishing engorgement of the cellular tissue in which the glands are embedded, if used too frequently and too continuously it may easily provoke inflammatory action. Experience does not seem to show that either it or the ointments of iodine are largely beneficial by any process of local absorption. The iodized collodion used by photographers is sometimes employed with apparent benefit, as it exerts a direct compression upon the part, while the iodides it contains are in a soluble form, and so far more suitable for absorption. It is also less unsightly, and does not harden and destroy the epidermis to so great an extent as do the other preparations of iodine. Whether the tincture or the ointment or a collodion containing iodine is used, they should not be applied too frequently, once in three or four days being often sufficient, especially in patients possessing delicate skins, as so many scrofulous sufferers do. The ointment of the iodide of lead rubbed into the part daily is highly recommended.

It is important that the part should be protected from cold and draughts, and where it is practicable the covering of the enlarged glands by a protective plaster is useful. A well-spread and flexible soap or lead plaster is often advantageous. Whether it stimulates the absorbents, and so favors the resolution of the tumor, or acts merely by protecting the parts from sudden changes in temperature and, above all, from unnecessary handling, it is unquestionably beneficial. If preferred, a belladonna plaster may be used, but care must be taken to see that the narcotic does not exert more than a local effect. Another useful application is a mixture of equal parts of belladonna and mercurial ointment, spread thickly on lint and kept in place by a bandage. The resort to these narcotics is especially indicated when pain is added to the other symptoms.

There is no use in poulticing enlarged glands until pus has formed and been given exit, as the formation of a large abscess is to be deprecated. Therefore, as soon as pus forms, a free opening should be made in such a position as will secure good drainage, and free suppuration should be encouraged by the use of warm dressings. It should not be forgotten, however, that while in adults a cure is to be hoped for chiefly by suppuration, in children there is a stronger tendency to resolution.

While thus pursuing general and local measures looking towards a cure, it is most important to see to it that the original cause of the trouble is not maintained. Therefore the patient should be carefully examined, to ascertain whether there is any peripheral disturbance which can be regarded as the original irritation. Especially in cases of cervical disease should the nares, pharynx, and tonsils be examined, and, if necessary, the proper treatment to effect a cure instituted.

Ordinarily the results of such local treatment as has been referred to are slow to manifest themselves, and they are too often not perceptible at all. We are, therefore, led to consider the adoption of more energetic and positive treatment. The measures which have grown markedly in favor of late years are excision, enucleation, and puncture with heated needles. All of them are undertaken with the idea of hastening the progress of the case and facilitating it by destruction of the diseased glands. All of them are justifiable measures in cases properly selected and discriminated.

Excision is especially indicated when but one or two glands are involved, when the relation to vital parts is not too intimate, and when the tumor is comparatively superficial and movable. The last requisite is most important. Such tumors as are suited for excision generally belong to the more slowly advancing cases, where there is little tendency to suppuration. As a general thing, there is little difficulty in the operation, the glands readily separating from their capsules when they are incised. But very often more glands may be involved than was at first expected, and no sooner is one gland removed than another presents itself from a greater depth. Owing, however, to the ease with which the gland and its capsule separate, it is often safe to shell out glands from greater depths than would be desirable if a close dissection were required. The risks of the operation are that when the skin and fascia are divided the gland may prove to have closer attachments than was anticipated. So intimate may these connections be by agglutination that the gland cannot be removed without the exercise of much force, which may risk the rupture of the gland and injury to neighboring structures. It is important to avoid the former mishap, lest the cheesy contents of the gland be disseminated through the tissues, while by the latter we may find ourselves confronted with injury to a large vessel or to the pleura or with the free opening of deep fascial planes. Indeed, the difficulty and danger of a deep dissection are so great that it is generally quite as well, if not better, to withdraw from the procedure when the conditions are found to be such as have just been described. Mr. Holmes once cut the axillary artery in removing a deep glandular mass in that region, thereby demonstrating the serious character of the obstacles to success, even in the most skilful hands. The advantages of excision in suitable cases are the speed and ease with which the gland is finally disposed of, and the insignificance of the linear scar left, which may be looked for, when antiseptic precautions are observed.

Enucleation or scooping is well adapted for many cases not suitable for excision, where there are adhesions to the skin or neighboring parts, or where sinuses lead from the surface to a diseased gland. The instrument used is a Volkmann's spoon or curette, by which the gland, or what remains of it, is scraped away. Access is had to the diseased gland either by a small incision through the skin and capsule of the gland, or by means of an existing sinus, and the substance of it is entirely scraped away. It especially suits cases where adhesions exist, and where, after long threatening, softening and cheesy degeneration have taken place. These glands ordinarily have very thick capsules, and there is on this account little risk of the spoon extending beyond the boundaries of the gland. The operation should be done with antiseptic precautions, and, if the cavity left is large, drainage should be secured.

Cautery-puncture has been strongly recommended by Treves, of London, and by some French surgeons. It is a simple and efficient means of hastening the progress of these tedious cases, and appears to be without drawback. A cautery-iron as large as a medium-sized catheter is heated red-hot and thrust through the skin and overlying tissues into the body of the gland, and made to traverse it in several directions without making a fresh puncture in the skin. The parts are steadied by the hand of the operator, and care is taken not to thrust the cautery beyond the boundaries of the gland. Should cheesy matter follow the withdrawal of the cautery, a poultice is applied ; otherwise a simple-cerate dressing is sufficient.

This mode of treatment is applicable to any case where the gland has attained a fair size,—say, one inch in diameter. It is easy of performance, and is followed by excellent results. When a gland which has not suppurated or become cheesy is subjected to this treatment, after temporary enlargement it shrinks up and tends to disappear, a cure being effected in a few weeks. When the gland contains much pus or cheesy material, the puncture made by the cautery affords free exit, while there is less risk of undermining of the skin from the infiltration of the surrounding tissues by the gland-contents than is the case where the spoon is resorted to. The evidence tends to show that, by exciting healthy action within the glands, cautery-puncture is superior to any other method of locally treating these cases, and is applicable to a greater number of them.

Electrolysis, and the injection into the body of the gland of some stimulating fluid, as acetic acid, iodine, or nitrate of silver, by means of a hypodermic syringe, are both useful plans of treatment, but not equal to that by cautery-puncture, either as to efficiency or as to the time required for a cure.

The same is true of the treatment by setons. The *rationale* is the same in all, while the results appear to be in favor of the hot iron.

Gland-abscesses should be opened early; and the weight of authority favors a very small incision. There is no reason why a large accumulation of pus should be allowed to take place. The rule to avoid pressure upon the gland-abscess is a good one,—as, in fact, it is in all abscesses. Should suppuration continue for a long time, the incision should be enlarged, and should, as is often the case, a diseased gland be found remaining, it may be touched with the hot iron. Sometimes the suppuration is kept up by a second deeper-lying gland which has communication with the abscess-cavity by a small opening. Until this gland is destroyed by inflammatory action the formation of pus will continue. It should, therefore, be searched for in obstinate cases, and subjected to the same treatment as its predecessor has received. When flaps of skin possessing but low vitality remain and interfere with the healing of the abscess, they should be removed; and this is best done by the actual cautery. In treating such cases the fact that rest is most important for the healing of wounds should not be forgotten, and the use of some appliance looking to this end should not be neglected. The constant motion of the neck may be much restrained by the use of a proper stock.

Thirty years ago the existence of scrofulous glands was considered antagonistic to the development of phthisis; but observation has abundantly demonstrated the fallacy of that theory. As proof of this fact, the following case is briefly narrated by way of conclusion:

Katy ——, aged four years, was admitted into the Children's Hospital, Philadelphia, under the writer's care, with symptoms of early hip-joint disease. After a year's rest and extension of the limb, she was sent out, apparently quite well. After some months' exposure to the neglect and unsanitary conditions of her home, she was readmitted, with a return of her old symptoms, and again was discharged benefited by treatment, only to be again received after an interval, with coxalgia and enlarged cervical glands. A large mass of the latter existed beneath the front of the jaw. She presented a marked strumous diathesis, and, after some delay, an examination was made with a view to interfering with the glands by operation. This was not considered justifiable, and in a few months tuberculous deposit was made out in both lungs and she succumbed to phthisis. The case was not unique, even in the experience of the writer, and is merely quoted from its bearing upon a theory once prevalent, but now exploded.

In concluding this paper it is both the duty and the pleasure of the author to acknowledge the many obligations he is under to the work on "Scrofula and its Gland-Diseases," by Frederick Treves, F.R.C.S., of the London Hospital, a volume which should be consulted by all who desire to make a more minute study of the subject.

PART V.

DISEASES OF THE MOUTH, TONGUE, AND JAWS.

DISEASES AND CARE OF THE TEETH.

By EDWIN T. DARBY, M.D., D.D.S.

In the consideration of the subject of the diseases of the teeth of children, the author cannot consistently confine himself exclusively to those of the temporary set, because many of the permanent teeth are erupted during childhood and are subject to the same diseased conditions and require much the same treatment.

Until recently the general practitioner has seemed to attach but little importance to the diseases of the temporary teeth, and less, if possible, has been done to encourage their preservation until such time as they should be replaced by the permanent ones.

That many nervous affections arise from diseases of the teeth in childhood there can be no doubt, and it seems eminently fitting that a treatise upon the diseases of children should embrace within its scope a chapter upon the teeth.

Standing at the very portal of the mouth and at the beginning of the alimentary canal, they are the chief agents in the mechanical part of the digestive function.

The teeth of the human subject consist of two sets: the first is called the temporary or deciduous, and the second the permanent. The temporary set consists of twenty teeth, ten in each jaw, and the time at which they are erupted is embraced between the sixth and the thirty-sixth month after birth. The teeth of the permanent set consist of thirty-two, and these are erupted between the sixth and the twentieth year, although the last of the series, the wisdom-teeth, so called, are sometimes delayed until a later period in life.

As early as the forty-seventh day of intra-uterine life there are indications of the development of the temporary teeth, but their eruption is usually postponed until about the sixth or seventh month after birth.

It occasionally happens that the central incisors of the lower jaw are present at birth, and cases are recorded where those of the upper jaw have also been present thus early. The notion that teeth so erupted should be extracted because of the pain caused the mother in the act of nursing her infant is as barbarous as it is unreasonable: there is no greater liability of the infant of a few hours old biting the nipple than of the infant of seven months, and few mothers would consent to weaning the child at seven months simply because it had erupted its incisor teeth.

The period of dentition is undoubtedly a trying one for the individual, and, although a physiological process which in the lower animal seems attended with little inconvenience, in the human subject it is regarded with great anxiety by the mother. Children that have been strong and healthy up to the period of dentition often droop and die, while the delicate or sickly ones pass through it with apparent impunity. It would seem a merciful provision of nature that a period of rest and opportunity for recuperation is afforded between the advent of the different classes of teeth; were it not for this, the tables of mortality would doubtless show a greater number of infantile deaths, although a study of them during the summer months is appalling.

The following formulas will show the names and number of the teeth of both the temporary and the permanent set, and the subjoined tables the time of their eruption:

TEMPORARY TEETH.

Incisors, $\frac{4}{4}$; cuspids, $\frac{2}{2}$; molar, $\frac{4}{4}$: 20.

PERMANENT TEETH.

Incisors, $\frac{4}{4}$; cuspids, $\frac{2}{2}$; bicuspids, $\frac{4}{4}$; molars, $\frac{6}{6}$: 32.

PERIOD OF ERUPTION OF TEMPORARY SET.

1. Central incisors	5th to 8th	month.
2. Lateral incisors	7th to 10th	“
3. First molars	12th to 16th	“
4. Cuspids	14th to 20th	“
5. Second molars	21st to 36th	“

The teeth of the lower jaw usually precede those of the upper by a few weeks.

PERIOD OF ERUPTION OF PERMANENT SET.

1. First molars	6th	year.
2. Central incisors, lower jaw	7th	“
3. Central incisors, upper jaw	8th	“
4. Lateral incisors, both jaws (lower preceding upper)	9th	“
5. First bicuspids	10th	“
6. Second bicuspids	11th	“
7. Cuspidati	12th	“
8. Second molars	12th to 13th	year.
9. Third molars (wisdom-teeth)	17th to 20th	“

From a study of the foregoing tables it will be seen that the child at six years of age has four of the permanent teeth, and that at twelve years of age he has twenty-eight teeth, all of which belong to the permanent series. The significance of this will be shown later on, when considering the care of the teeth.

IRREGULARITIES AND ABNORMALITIES.

Irregularities, either as regards size, number, or position, are not so frequently met with in the temporary as in the permanent series, although they are not so rare as is often supposed by the casual observer.

Abnormality in size is not often attended with any unfavorable conditions, provided the arch is sufficiently large to contain all the teeth without undue crowding. The central incisors of the upper jaw are sometimes a third broader than normal, and the second temporary molars are frequently so large as to resemble the first permanent molars. The lateral incisor and the cuspid of the lower jaw are sometimes found to be united in one large crown, although a line of confluence is usually recognizable in the enamel on its labial aspect.

The abnormal size of the molars is rather favorable than otherwise, because, in addition to the greater masticating surface which they present, they hold a larger space in reserve for their successors (the bicuspids), and thus prevent undue crowding of these teeth, a condition which is frequently attended by the most serious consequences.

Irregularity as to Number.—Deficiency or excess in number is not so frequently seen in the temporary as in the permanent teeth, although it occasionally happens that one or two teeth are wanting, and occasionally one or two teeth above the number are present.

There are a few well-authenticated cases on record where neither temporary nor permanent teeth have been erupted, and the individuals have gone through life edentulous.

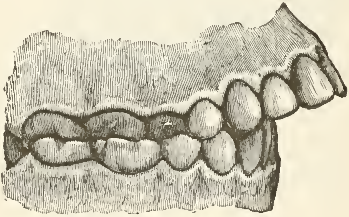
The author has in his collection casts of the upper and lower jaws of a lad seventeen years of age in which there are but two molars, both in the upper jaw. He had no temporary teeth in either jaw, and until he was twelve years of age had not erupted a single tooth. The absence of the lateral incisors of the upper jaw has frequently been observed, and both central and lateral incisors of the lower jaw are sometimes found wanting. An additional lateral incisor is occasionally seen in the upper jaw, a peculiarity which may manifest itself in several children of the same family. When teeth above the regular number are found in the temporary set, they usually partake of the shape and characteristics of the other teeth, whereas in the permanent series the supernumerary teeth are not only in excess of a given type, but are also triangular or cone-shaped and developed in abnormal positions in the jaw, behind the upper incisors, or insinuating themselves between the molars of the same jaw.

Irregularity as to Position.—It may be said that when teeth are normally arranged in the jaw they describe two parabolic curves, the upper being the larger and closing slightly over those of the lower. Teeth thus arranged in a well-developed jaw may be said to be regular. Such regularity is more frequently observed in the temporary than in the permanent set, and yet deviations from this are frequently met with in those of the temporary series. The cause of this deviation may be hereditary or acquired, and may be so slight as to escape the notice of the casual observer, or so great as to amount to actual deformity of the facial expression.

One of the most common deviations from a normal arrangement is to be found in the lower jaw, the teeth of which, instead of closing behind those of the upper, meet upon the cutting edges or close outside of those of the upper jaw. Such an occlusion has been denominated prognathous. It is more frequently than otherwise an hereditary condition, and is often observed in several members of the same family. Being of congenital origin, little is usually done to remedy the evil during childhood or until after the permanent teeth begin to make their appearance.

Another type of irregularity is sometimes met with in the upper jaw, and is the result of the habit of thumb- or finger-sucking. (See Fig. 1.) The incisors of the upper jaw are prominent, protruding much beyond those of the lower jaw. The jaw is narrowed laterally and presents a contracted appearance across the hard palate. The teeth of the lower jaw are forced inward, because of the thumb or finger resting upon them in the act of sucking. Whenever this habit is acquired in infancy, it should be corrected as early as possible, for, if retained beyond the period when the permanent teeth take their position in the jaws, they too may be forced outward and the lower ones inward until an unsightly irregularity of the teeth of both jaws will be produced, thus impairing speech and rendering the biting of food difficult. The author has in his own practice a young man of seventeen years who still continues the habit of thumb-sucking and says he is unable to break himself of it,—he awakes in the night to find his thumb in his mouth; the deformity in the case mentioned is great, the incisors of the upper jaw protruding beyond those of the lower to an extent equal to the diameter of the thumb.

FIG. 1.

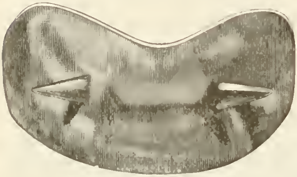


The habit may be broken in infancy by wrapping the thumb or finger with muslin saturated with aloes or some harmless preparation disagreeable to the taste; children can sometimes be shamed out of the habit to such an extent that they will refrain from it in the presence of others, but when alone, or when darkness screens them from observation, they will renew the practice. Lip-sucking is another habit which often causes depression of the lower incisors. The habit is acquired by drawing the lower lip into the mouth, and by its pressure upon the teeth they are forced inward to such an extent that deformity is the result. The space for the posterior teeth is greatly curtailed, and extraction of one or more teeth is required to make room for all in the arch. If the child cannot be broken of the habit otherwise, a fixture should be placed between the teeth and the lip which will make it impossible for the lip to be drawn into the mouth.

Mouth-breathing is another habit which often results in the lateral contraction of the upper jaw and produces irregularity of the teeth. When this habit is formed as the result of some obstruction in the nasal air-passages, surgical treatment is required; frequently, however, it is acquired when no defect in these parts exists.

Perhaps the most effectual method of breaking this habit is that of the Indian mother, who bandages the mouth of her infant and compels it to breathe through its nostrils or else not breathe at all. The author was recently shown a very ingenious device for preventing mouth-breathing, used by Dr. F. B. Darby, of Elmira, New York. It consists of a vulcanized rubber plate which encircles the labial faces of the teeth of both jaws and is held in position by a band around the head. It is to be worn at night, and while the mouth is at rest during the day. Its presence in the mouth makes mouth-breathing more difficult than nasal breathing, and thus the habit is corrected.

FIG. 2.



Rubber plate.

DENTAL CARIES.

Caries of the teeth has probably existed in all ages of the world; at least it may be said to be as extended as mankind. All nations of the earth seem to have suffered from its ravages, although not to the same extent. The remains found in ancient tombs show unmistakable evidence of its existence, and recent explorations have demonstrated beyond the possibility of a doubt the efforts to combat its influence or to repair the injury caused by its ravages. Within the past year or two, specimens of ancient dentistry have been taken from Etruscan tombs, and carious teeth have been found in the mouths of embalmed mummies that have for forty centuries been buried in the sands of Egypt: hence the theory that caries of the teeth is a modern disease and the result of civilization is a false one. Prehistoric man was doubtless familiar with all the pangs of toothache, without the means of relief which are so common at the present day.

In the consideration of the etiology of dental caries the author can give but an outline of the various theories which from time to time have been promulgated; indeed, the limited space allotted will hardly admit of an intelligent *résumé* of this branch of the subject. Interesting as these theories are, in the light of recent investigation they seem crude indeed, but, with the imperfect facilities for microscopic observation which existed a century or even half a century ago, it is not strange that so much of error and so little of truth found its way into the minds and teachings of the early histologists.

Hippocrates, who was a humoral pathologist, attributed the cause of caries to the bad condition of the humors of the body, and for a thousand years this belief was prevalent. Fauchard believed in the worm hypothesis, and sought in vain in carious and dead teeth for the worm that produced the disease. Bourdett thought that caries was the result of putrefying dental vessels supplying the teeth, and that the disease arose from an internal cause.

John Hunter, although a careful observer, has not shown his customary astuteness in relation to the cause of dental caries: he confounds caries of the teeth with gangrene or mortification, and believes that it is the result of inflammatory conditions.

Mr. Fox (1806) regarded caries as the result of inflammation of the lining membrane of the pulp-chamber (membrana eboris); he also believed in the internal theory of caries.

Mr. Bell (1829) also believed in the inflammatory theory of dental caries, but thought that portion farthest from the pulp least able to resist the process of mortification. Koecker evidently endorsed much of the views of Bell, but recognized a chemical process which he thought played an important part in the destruction of tooth-substance. He departed somewhat from the views of his contemporaries in regard to the seat of caries being an internal one, and asserted that it never penetrated the pulp without first manifesting itself upon the surface of that organ. The inflammatory theory of caries was stubbornly contradicted by Harris, Robertson, and Rignard as early as 1830, because they had observed that human teeth mounted upon natural roots or attached to plates were as liable to decay as those that retained their vitality. Rignard in 1838 defined caries as "destruction by decomposition," and endeavored to prove his theory by a series of experiments, which consisted of loosely-fitted bands of metal about the teeth, thus affording a lodgement for particles of food, which, being held in contact with the surface of the tooth and becoming decomposed, generated an acid which decalcified the enamel of the tooth thus enclosed.

Mr. Tomes, although an eminent histologist and a close microscopic observer, seems to have been led by his observations to attribute dental caries to a vital or chemico-vital cause, and fails to recognize the important part which fermentative processes exert in the destruction of tooth-structure.

Magitot[1] evidently adheres with great pertinacity to the chemical theory of caries. He seems to be of the opinion that caries of tooth-structure identical in form can be produced in the laboratory by the aid of mineral and vegetable acids, an opinion which in the light of recent investigation would be strongly contested unless he admitted the active part which fermentation and micro-organisms play in the production of dental caries.

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[1] Treatise on Dental Caries, by Dr. E. Magitot, translated by Dr. Thomas Chandler.

Dr. Watt[1] promulgated the mineral-acid theory of caries, but, while his theories were most ingeniously presented, they failed to meet the endorsement of the profession at large. Leber and Rottenstein[2] were the first to recognize the active part which micro-organisms play in the production of dental caries, and, although others have gone further and singled out the nature of the microbes which seem most active in the production of the disease under consideration, there is no doubt that these observers were the first to call attention to their existence in carious teeth. They accept the chemical theory in so far as that the enamel must first be destroyed by acid, but when that has been decalcified a parasitic plant or fungus, which they term leptothrix buccalis, penetrates the dentinal tubes and, by distending and enlarging them, renders the process of disintegration much more rapid. The theory of Leber and Rottenstein was thoroughly endorsed by Messrs. Mills and Underwood, of London, and the result of their work in the same direction was given to the Section on Oral and Dental Surgery at the meeting of the International Medical Congress held in London in 1881. These gentlemen, by a careful and painstaking series of experiments, demonstrated, beyond the possibility of a doubt, the presence of micro-organisms in the dentinal tubes, and gave to the scientific world the most positive evidence that these organisms play a very important part in the production of caries.

Dr. W. D. Miller has gone still further than Messrs. Mills and Underwood, and has told us the character of the microbes, and by a series of cultures has been able to reproduce them to an unlimited extent and study their life-histories. His experiments have been most carefully performed, and are worthy of close study.[3] By his culture-mediums he is able to take a fragment of decay from the bottom of a carious tooth and produce caries in sound teeth of identically the same nature as that found in the mouth. It is unquestionably true that most mouths contain myriads of the micro-organisms, and all that is needed for the production of caries is a favorable lodgement or sheltered place where fermentation may go on uninterruptedly; such places are to be found in the interstices between the teeth, in the grooves or fissures upon the masticating surfaces, or at such points as the brush does not reach or the tongue cleanse.

The enamel is destroyed by the acid of fermentation, and the micro-organisms, being ever present, penetrate at once the tubes of the dentine. Dr. Miller has found that the micro-organisms secrete lactic acid, which is sufficient to dissolve the inorganic constituents of the tooth, and it would seem that all the conditions favorable to the production of caries are present. The author would be glad to give in detail the series of experiments which Dr. Miller so carefully made, but he must refer the reader to his publications.

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[1] Chemical Essays.

[2] Dental Caries and its Causes: an Investigation into the Influence of Fungi in the Destruction of the Teeth, by Drs. Leber and Rottenstein, 1867.

[3] The experiments of Dr. Miller were published in the *Independent Practitioner* of February, March, and May, 1884, and May and June, 1885. See also article in System of Dentistry, vol. i. pp. 791 to 826.

Phenomena of Caries.—From what has been previously said, the reader will doubtless infer that little reliance is placed upon the internal theory of caries. Unlike caries of the osseous system, the decay of the teeth invariably begins from without and progresses inward towards the pulp or central portion of the tooth. It does not attack clean or smooth surfaces, but such points as are rough or imperfectly developed, or places that retain food or the secretions in contact with them.

The points most frequently attacked by caries are the fissures upon the masticating surfaces of the bicuspids and molars, the grooves and depressions upon the palatine surfaces of the incisors and the buccal surfaces of the molars, and the proximate surfaces where the teeth are in contact or so nearly in contact that particles of food are retained by them. Another frequent seat of caries is at or near the neck of the tooth, where the enamel joins the cement, especially if there be a little recession of the gum.

The appearance of caries varies with the structure of the tooth; in teeth that are hard and of good quality the decay is usually dark in color, whereas in teeth that are soft or poor in structure it may vary from yellow or light brown to almost white. The color of decay may, therefore, be taken as a guide to the quality of the teeth: white decay is always rapid in its progress, and dark decay is usually slow.

An examination of the grooves or fissures upon the masticating surfaces of the molars will reveal, when dry, either dark stain or a chalky appearance. Not all fissures which are dark are necessarily carious, because they are sometimes filled with stain, whereas all fissures which present the white or chalky appearance will be found by probing to be carious, and oftentimes the disease has penetrated to a considerable extent into the substance of the dentine before the enamel shows signs of breaking down. A careful examination of a tooth of average density in which caries is progressing will show a zone of discoloration, cone-shaped in appearance, with its apex looking towards the pulp and its base towards the outer surface of the tooth. It will be found that caries progresses most frequently in a line with the dentinal tubes, and in proportion as the teeth are hard this rule will hold good.

In teeth that are of poor structure little discoloration is observable, and the carious portion especially of the enamel is whiter than the tooth surrounding it. In teeth of this character the decay is less likely to follow the dentinal tubes, but seems to spread out underneath the enamel, and is sometimes called "spreading" caries. This type of caries is often seen in the wisdom-teeth, which, from want of space for development or because of premature eruption, are poor in structure and succumb to caries when once attacked.

Predisposing Causes.—While it is true that all teeth are liable to caries, it is also true that some teeth do not yield to the disease. It occasionally happens that an individual attains a good old age without a single carious tooth, but such instances are rare and are always the subject of remark because of their rarity. It might be supposed that people living under the same climatic conditions, eating the same food, and observing the same laws of health would be equally exempt from caries, or, to put it differently, that teeth subjected to the same unfavorable conditions would yield alike to disease; but there are certain predisposing conditions which render some teeth more liable to caries than others.

First among these may be mentioned imperfect formation, and this imperfection may be twofold, either as relates to structure or as relates to form. The tooth that is soft or wanting in lime salts must of necessity yield the more readily to the action of acids and microbes, and, having yielded, must the sooner succumb to their destroying influences. So also the tooth that is imperfect in form, in which the enamel is not only poor in quality but has blemishes, such as pits or depressions, is the more liable to attack in these weak places by the acid which is the result of fermentation of food held in contact with them. The shapes of teeth have more to do with their immunity from caries than is generally supposed. A tooth with long cusps and correspondingly deep fissures is specially favorable to the retention of fermentable substances; so also the long teeth which present large surfaces in contact with adjoining teeth are most liable to disease from the causes above referred to, and this leads to the consideration of another predisposing cause,—viz., contact.

Teeth that stand alone or are considerably apart in the mouth do not decay upon their proximate surfaces, but when in contact, or nearly so, they offer favorable lodging-places for particles of food and the secretions which by the process of fermentation and the generation of acid render such surfaces almost sure to be attacked by caries.

Hereditary influences may be classed among the predisposing causes of caries. It does not follow, because one or both parents have suffered from caries of the teeth, that the offspring will also suffer from the same disease; but there is a strong tendency for like to beget like, and it is the transmission of similar conditions, and not of special disease, that we recognize as hereditary.

If the child inherits the imperfect form, the imperfect structure, and the crowded condition of the teeth which the parent had, and in addition thereto inherits the same abnormal secretions of the mouth, the chances are largely in favor of the same dental lesions; and yet by superior care and watchfulness the adverse conditions may be controlled and the special disease averted.

It is unnecessary to say that in mouths where the secretions are unfavorable to fermentation and the teeth kept scrupulously clean upon all surfaces, even teeth of inferior quality will resist decay, whereas teeth of the best quality surrounded by secretions susceptible to fermentation will probably yield to the bad influences notwithstanding their superior quality. Caries does not seem dependent upon an acid or alkaline condition of the fluids of the mouth, but progresses with the same apparent rapidity under either condition. It is not so much the general character of the oral secretions as it is the chemical character at the seat of disease, and this is determined by the processes of decomposition and fermentation.

Progress of Caries.—Caries in its earlier stages may elude the notice of the inexperienced observer. The disease may be recognized by an opaque spot upon the surface of the enamel, or upon teeth of harder structure there may be a brown or at times an almost black appearance to that portion of the tooth in which the disease is progressing. Caries is attended with no sensation until it has penetrated the enamel; in fact, in teeth of dense structure it may progress to a considerable extent into the substance of the dentine before pain is produced.

Sensation to thermal changes is among the first indications of its presence; a slight pain is produced when hot or cold fluids are taken into the mouth or when sweets are brought in contact with it. The pain may be but momentary, but, as the disease progresses and the dentinal fibres become exposed to the secretions of the mouth or to atmospheric conditions, more or less pain is experienced. Although there is a great similarity in the diseases of the temporary and those of the permanent teeth, the course of treatment is not always identical, and, as the present work relates to such diseases as are peculiar to children, it may be well to distinguish between the temporary and the permanent teeth and consider first the treatment indicated for those of the temporary series.

It is not uncommon for the temporary teeth to be attacked by caries soon after their eruption, and seldom do they subserve the purpose for which they are intended before some of them at least yield to the ravages of decay.

Various theories have been advanced to account for the wide-spread disease of the teeth in children at the present day, and, although it may be impossible to fix upon a single cause which would be a satisfactory solution of the problem, it is reasonable to suppose that the character of the oral secretions and the nature of the food have much to do with the prevalence of caries in the teeth of the very young.

The author has observed that children raised upon condensed milk or those who partake of food into which sugar enters largely are apt to suffer greatly from carious teeth. The habit of giving the young child a "sugar-teat" to keep it quiet is certainly a pernicious one, because the secretions would be found acid a great part of the time. Sugar as sugar does not destroy tooth-structure, but when it enters into combination with the secretions of the mouth its character is almost instantly changed and acid is the result.

The points most frequently attacked by caries are the fissures upon the masticating surfaces of the molars and the proximate surfaces of the molars and incisors; but it is not uncommon to find the labial and buccal surfaces of the teeth also the seat of decay.

The caries of childhood is often very rapid in its progress, doubtless owing in part to the insufficient care in cleansing, and the vitiated condition of the fluids of the mouth.

As soon as caries has penetrated the enamel and the dentinal fibrils become exposed, the child is conscious of pain if anything sweet be taken into the mouth, and toothache of a severe character is sometimes experienced although there be no exposure of the pulp. The pain may be allayed by putting into the cavity of decay a little bicarbonate of sodium or a small pellet of cotton saturated with the oil of cloves, creasote, camphor, laudanum, or chloroform. A little sulphate or acetate of morphine will also give almost instant relief.

When caries is found to have commenced in the mouth of the child, it is well to have it visit a dentist, that all cavities may be filled while yet small and before the operation becomes a painful one. Some of the plastic materials are best suited for children's teeth, such as gutta-percha, phosphate of zinc, or an alloy for the posterior teeth. Such treatment will not only prevent a great amount of suffering, but will also tend to preserve the teeth until such time as nature shall cast them off by absorption of their roots.

If decay be not arrested in its earlier stages, it will progress deeper and deeper into the substance of the dentine, and finally the pulp will become exposed.

PULPITIS.

Pulpitis, as its name implies, is an inflammation of the dental pulp. In the central portion of each tooth there resides a mucoid gelatinous matrix containing blood-vessels and nerves in abundance, to which the name of tooth-pulp has been given. It is the remains of the formative dentinal organ, and is the internal source of nutrition for each tooth. When from caries or other causes this becomes exposed and irritated, congestion and inflammation supervene.

Fragments of carious dentine or enamel or particles of food are sometimes forced into the pulp-chamber or encroach upon the pulp to such an extent as to cause intense pain; these should be gently removed, the cavity washed with warm or tepid water, and, if possible, the congested pulp slightly depleted by touching with needle-pointed instruments, after which a dressing of dilute carbolic acid, oil of cloves, or creasote may be applied.

A few applications of carbolic acid are usually sufficient to devitalize the pulp in the temporary teeth. Arsenious acid, which is largely used for the devitalization of the pulps in the permanent teeth, is contra-indicated in those of the temporary set, owing to the greater vascularity of the temporary teeth and the open foramen in all teeth in which absorption of the roots has commenced.

After the pulp has been devitalized and removed, the pulp-chamber, canals, and cavity of decay may be filled with some plastic material, as above indicated.

PERICEMENTITIS.

Pericementitis is an inflammation of the membrane lining the socket and covering the root of the tooth. It may result from blows, from undue pressure, or from any foreign substance coming in contact with or remaining against it, but the most frequent cause of pericementitis is the death of the pulp. If from any cause the pulp of a tooth becomes devitalized and is allowed to remain in the tooth, it decomposes, and portions of it or the gases arising therefrom are liable to be forced through the apical foramen, setting up by septic influence irritation and inflammation in the pericemental covering at the apex of the root.

The symptoms of pericementitis in its earlier stages are, first, an uneasy feeling in the tooth and a disposition to press the tooth of the opposite jaw upon it, but soon after the same degree of pressure causes pain, and later on the slightest pressure of the finger-tips or tongue causes intense pain. The tooth is elongated, and when the jaw is closed it seems the first to touch. As the disease progresses, swelling of the parts ensues, and frequently great distention and disfiguration of the face are observed. In the earlier stages it will often yield to continuous applications of cold, either in the form of ice held upon the adjacent parts or by means of rhigolene or ether spray. Depletion is often of service, and may be accomplished by lancing the gum freely near the point of disease or by applying leeches to the gum.

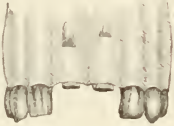
Counter-irritation, by painting the gum with strong tincture of iodine or by using equal parts of tincture of iodine and tincture of aconite root upon the gum, will often give relief. Small capsicum plasters applied to the gum over the root of the tooth will sometimes be of service. If there be failure to arrest the irritation in the earlier stages, suppuration will ensue, and then we shall have as the result—

ALVEOLAR ABSCESS.

This may be defined as the suppurative stage of pericementitis. An abscess under any conditions is painful, but when surrounded by bony walls, with no escape for the pus, the pain is intense. When all efforts to prevent suppuration or arrest the disease in its earlier stages have failed, it is desirable to hasten the discharge and evacuate the contents of the sac as speedily as possible. To accomplish this, hot applications or fomentations should be applied to the gum immediately over the root of the tooth. Equal parts of milk and water heated to a temperature of 105° F. and held in the mouth will often give relief from pain and hasten the discharge. Comfort is often obtained by holding a warm decoction of poppy and chamomile in the mouth. If the abscess be upon an upper tooth, there is no objection to external poultices; but when situated in the lower jaw, such a procedure is contra-indicated, as there is always danger of drawing the pus to the surface, thus leaving an unsightly cicatrix. Should there be indications of an external discharge of the pus, a compress should be applied at once, thus encouraging the pus to burrow through at some other point in the cavity of the mouth. Alveolar abscess, both acute and chronic, is very common among children. It seems to be the natural sequence of neglected caries. The pulp becomes exposed, and dies either from inflammation or strangulation or as the result of a medication; if it be not removed and the tooth filled, decomposition ensues, and the septic matter finds its way to the pericementum, with the result above mentioned.

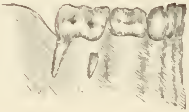
An acute attack of alveolar abscess may run its course in twelve hours, or it may be of several days' duration. The swelling gradually subsides, the tooth resumes its natural position in the socket, and the health of the part returns, but more frequently a sinus remains, through which there is an occasional or constant discharge from the old sac. This chronic condition often results in absorption of the anterior plate of the alveolar process near the affected tooth, and frequently the ends of the roots are left without bony or even gum covering, as seen in Figs. 3 and 4. Whenever this condition presents, the ends of the roots should be excised with cutting forceps and the remaining portions nicely smoothed with a file or wheel revolved by the dental engine. If these projecting roots be allowed to remain, they often cause ulceration upon the inner surface of the lips and cheeks and are the cause of much distress to the individual. With the death of the pulp of a tooth, the physiological process of absorption of its roots ceases, and such teeth either are exfoliated *en masse* or must be extracted when their successors, the permanent teeth, begin to make their appearance.

FIG. 3.



Upper jaw with portions of the roots of temporary central incisors exposed.

FIG. 4.



Lower jaw with lower second temporary molar having one root fully and one root partially exposed.

THE CARE OF THE TEETH.

The care of the teeth should begin in infancy. As soon as the temporary teeth begin to make their appearance, they should receive the attention of the mother or nurse, while the child is still young; they may be cleansed daily with a soft linen or muslin rag, but as soon as the teeth are all present in the mouth a small soft tooth-brush should be used upon them once or twice daily. The child can early be taught the use of the brush, and not unfrequently a child two or three years of age will become so habituated to its use that it will not feel comfortable until its teeth have been properly cleansed. Stains upon the teeth are not only unsightly even in the mouth of a child, but they may be of such a character as to be injurious to the teeth. The green or brown stains which are sometimes seen upon the labial surfaces of the teeth are usually the result of an abnormal condition of the oral secretions, and a microscopic examination will often show them to be of fungous origin. These stains when present should be polished off, either with a fine powder of precipitated carbonate of lime or with a pine stick dipped in fine pumice-stone.

Salivary calculus—or tartar, as it is commonly called—is found upon the teeth of all persons, although not to the same extent. It may be of various colors, either white, yellow, or brown or almost black. It is a calcareous deposit from the saliva, and, where mixed with mucus and other substances found in the mouth, precipitates upon the teeth in greater or less quantities. While salivary calculus does not injure the teeth themselves, it has a very destructive action upon the gums, causing them to become congested and inflamed and to recede from around the necks of the teeth, and the teeth themselves to loosen and fall out. Whenever it is found in the smallest quantities, it should be promptly removed, and the surface of the tooth to which it was attached thoroughly smoothed. The importance of thoroughly brushing all surfaces of the teeth cannot be over-estimated. Comparatively few adults, much less children, perform this part of their toilet with anything approaching thoroughness: they simply cleanse with the brush the labial surfaces of the teeth, leaving those surfaces which are more difficult to reach almost or quite untouched, and it is in these hidden places that deposits are found and that caries occurs. The young child should be taught the importance of picking the teeth with a quill pick after each meal, to insure the removal of all particles of food which may have lodged in the interstices between the teeth, where, if allowed to remain, fermentation ensues and decay is almost sure to follow. The use of floss silk nicely waxed and passed between the teeth after each meal and always before retiring at night cannot be too highly recommended.

Dentifrices composed of precipitated carbonate of lime, pulverized orris-root, pulverized myrrh, and cinchona bark, with the addition of a little pure Castile soap, may be used once or twice daily, with the best results. Nothing adds more to the appearance of a child's face than a row of pearly-white teeth, and nothing detracts more than a mouth full of diseased, discolored ones. To attain the former a little care only is necessary in early infancy, because when the habit of caring for the teeth is once acquired it continues throughout life.

The temporary set of teeth are frequently the seat of caries: hence the importance of early dental treatment. The child of two years should be taken to the dentist, that its teeth may be carefully examined, and if decay has begun on any of the teeth they should be treated as indicated elsewhere. Periodical visits at intervals of six months should be made to the dentist, to insure freedom from pain and the retention of the temporary teeth until they are displaced by the advent of the permanent set.

At the sixth year of age the first teeth of the permanent set make their appearance in the jaw. These are the four first molars, and their position is posterior to the molars of the temporary set. Great ignorance, even among people of more than ordinary intelligence, exists in regard to these teeth. The belief is general that all teeth erupted in childhood belong to the temporary set, and it is not uncommon for these molars of the permanent set to be allowed to decay until all hope of saving them is doubtful or impossible before the dentist is consulted. They are the largest teeth of the dental series, and are situated where they perform the most important part of the masticating function, and their preservation throughout life is of paramount importance. Every mother and every person who has the guardianship of children should bear in mind, when the number of teeth in the mouth of a child exceeds twenty (ten in each jaw), that all in excess of this number are teeth belonging to the permanent series and should be watched with the greatest care. The loss of a single permanent tooth in early life may not necessarily be attended with inconvenience or injury, but more frequently than otherwise such a loss impairs mastication, mars the harmony of facial expression, and destroys the symmetry of the whole face.

CONGENITAL DEFECTS AND DEFORMITIES

OF THE

FACE, LIPS, MOUTH, TONGUE, AND JAWS.

By ROSWELL PARK, A.M., M.D.

There can be no intelligent and comprehensive appreciation of the subject of congenital defects of the face and mouth without a brief reference to the embryology of the parts.

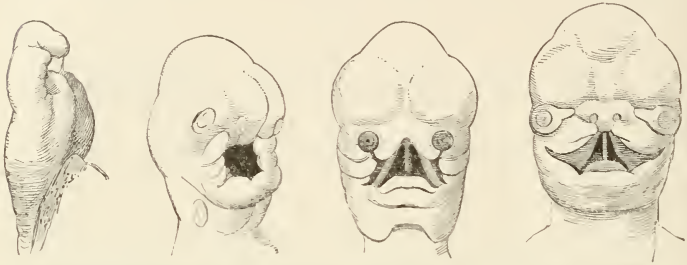
On the anterior surface of the rudimentary cranium appear four pairs of clefts, while between them the tissue thickens into protuberances which later become processes and expand to meet their fellows of the opposite side. The first of these arches, the pre-oral or maxillary, unites with the fronto-nasal process which projects downward from what is to be the frontal bone. This process is in reality a triple one, whose central part is called the mid-frontal process and from which the nasal septum is developed. The lateral plates of the naso-frontal process separate from the mid-frontal, and by their divergence form the primary nasal pits or fossæ; they shut off these fossæ, while in them are developed the lateral masses of the ethmoid and the lachrymal bones. By the union of these latter with the mid-frontal portion at certain points are produced the intermaxillary bone and the philtrum or lunula,—the central part of the upper lip.

The maxillary processes arise farther back than the fronto-nasal. They descend a short distance, from the outer wall of the orbit and the malar bone, then turn inward, and, meeting the lateral portion of the mid-frontal process, form the floor of the orbit and shut it off; then, passing downward and inward, they meet the mid-frontal process, and with it complete the alveolar arch, the upper maxilla, and part of the cheek. The palate is formed by development inward of the inner portion of each half of the maxillary arch thus formed.

The post-oral or branchial arches are five in number, of which only the first interests us here, since by the blending of the two inferior maxillary processes there is formed the lower jaw, which constitutes this first arch. Between the pre-oral or maxillary arch and the post-oral or mandibular the mouth is formed: its walls consist of mesoblastic tissue lined by a continuation inward of the epiblast.

When the complicated arrangement of the offshoots from the various processes has been thus pointed out, it will be much easier to understand the various deformities that may arise from failure to unite, or from excess or deficiency of development.

FIG. 1.



Development of the nose and mouth, showing the mid-frontal, nasal, and maxillary processes, and the formation of the maxillary arches. (Coste.)

In general, one can say, with Trendelenburg, that the process of amalgamation of these parts is easily disturbed, and that defects apparently occur most frequently between the frontal and superior maxillary processes. The lower border of the superior maxillary process should fuse with the upper margin of the first branchial: in this way the cheek is formed. The relation of the superior maxillary process to the frontal process, by disturbance of which the orbito-nasal fissures are formed, is quite complicated, inasmuch as the frontal process projects itself downward between the two maxillary processes, and thus there are greater possibilities for irregularities.

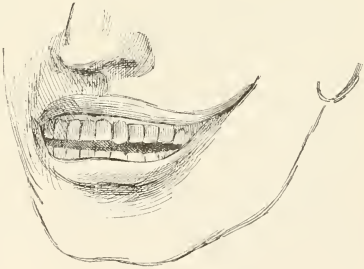
In the mouth the tongue is built up of three parts, the two lateral being produced from the lateral portions of the first pharyngeal arch, and the third projecting upward from the middle of the second and third pharyngeal arches to unite with the other two: the three together at first form a sort of tubercle in the floor of the rudimentary mouth. The hard and soft palates are formed from the median borders of the superior maxillary processes: by their amalgamation the closure of the fissure between these two begins at some distance back of the intermaxillary bone.

The disturbances of development which lead to these congenital deformities of the face all have their beginnings very early in fœtal life, and are simple failures to complete that which was contemplated in the development of the individual, so far, at least, as the defects and fissures are concerned. Sometimes there is excess of development, in which case we have deformities of another class. Lateral or bilateral fissures of the lips, of the alveolar border of the hard palate or of the soft, can be combined with one another in various ways. Discussions are not of enough value to deserve a place here; still, almost all authors agree in believing that lateral fissures of these parts occur more frequently on the left side than on the right. Some of the worst defects are produced by irregular or bizarre development of the mid-frontal process. Fusion with the maxillary process may have been disturbed, or occasionally the vomer is practically lacking; sometimes the cutaneous septum is lacking, in which case we have the appearance of a single nostril; usually in such cases there is a palatal or an alveolar defect; frequently both nasal bones also are wanting, and not infrequently there are serious defects in other parts of the bony skull or even of the brain itself. In a case reported by Engel, the walls of the nose and the nasal and the intermaxillary bones were lacking, there were rudimentary incisor teeth, both jaws were badly deformed, the space between the orbits was reduced to almost nothing, and other defects were noticed.

Median hare-lip is certainly extremely rare in man, though comparatively frequent in other mammals and normal in many rodents. As the median process develops to form the nose, two round prominences appear at each angle; they are the globular processes of His, and give rise to the alæ of the nose and the premaxillæ; they are later joined by the lateral pieces to complete the lip. These globular processes almost invariably unite in man, but are not so constantly joined by the lateral portions; consequently the vastly greater frequency of hare-lip on one side or the other of the middle line.

When we have failure to unite between the external frontal and the superior maxillary processes, we have to deal with oblique fissures of the face. These fissures usually begin on the free border of the upper lip about at the point where hare-lip is most commonly met with, rarely at the angle of the mouth. They are usually directed towards the ear or the external angle of the orbit, sometimes opening by a coloboma of the lower lid. These oblique facial fissures may be lateral or bilateral: they involve generally only the soft parts of the face, rarely with some congenital bony defect beneath. If this bony defect be in the hard palate the case is quite similar to one of combined hare-lip and cleft palate.

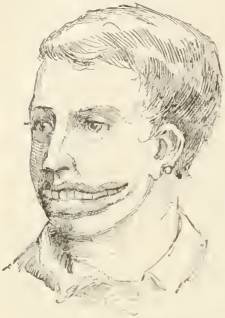
Fig. 2.



Oblique fissure of the face.

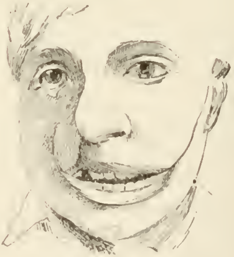
When we divide the fissures which may affect the upper lip upon embryological grounds, we have the following: hare-lip is met with between the philtrum—that is, the lower end of the middle frontal process—and the middle wedge-shaped strip which corresponds to Albrecht's interlabium externum, while between this part and the portion which is developed from the superior maxillary process we meet with the oblique facial fissures already spoken of. When the fusion between the superior maxillary process and the first pharyngeal arch has failed, there results horizontal fissure of the face or of the cheek proper; which also may be met with on one side or on both. For the most part these begin at the angle of the mouth and pass outward and a little upward, less often a little downward, in the general direction towards the angle of the lower jaw. The fissure may be lined with mucous membrane corresponding to the vermilion border of the lip, or may be covered with integument: it constitutes an extension of the mouth and forms one variety of macrostoma. A little tubercle on one of its borders often marks the insertion-point of the defective orbicularis oris. In extent it varies widely. In two cases of Fergusson's it reached as far as the masseter. In a case of the elder Langenbeck, it extended to the border of the jaw. Von Lesser described a double fissure of this kind extending on the left side nearly to the angle of the jaw, and on the right side to the point between the condyloid and coronoid processes. Most of these cases are combined with a rudimentary development either of the entire lower jaw or of that half on the side affected. C. O. Weber and Pelvet observed a combination of horizontal fissure on one side with oblique fissure on the other, and Fergusson reported a combination of fissure of the cheek on one side with hare-lip on the other. Naturally, an extensive case of this kind is an impediment to the holding of fluids in the mouth, and may render the act of swallowing impossible.

FIG. 3.



Horizontal fissure of the face.

FIG. 4.



Complicated facial fissure.

When the superior maxillary processes have united to too great an extent with the first pharyngeal arches, we have a deformity by which the mouth is made too small; in other words, this constitutes one form of microstoma. The oral opening is thereby reduced to a small round one, which may be so narrow as scarcely to admit the tip of the little finger. In such cases the lower jaw is too small, and there are usually accompanying deformities of other parts. Failure to unite between the parts of the first pharyngeal arches is known, in consequence of which we have median fissures of the lower lip, of the lower jaw, or of the tongue. These are very rare. Bouisson described in 1840 a complete fissure of the lower lip which corresponded precisely to an ordinary hare-lip. Ribely has operated upon a seven-year-old boy with a similar fissure which extended to the chin. A brother of this boy, by the way, had the usual form of fissure of the upper lip.

Aside from these deformities, which are simple defects, we have others which are either a complication of defects with excesses or else are to be classed with the teratomata, a class of neoplastic malformations about which we are yet sadly ignorant. A study of these masses would lead us away from the prime object of this article. The following will serve as examples of anomalous and bizarre arrangements such as may be met with about these parts.

Luschka noted in the skull of a female infant, dying shortly after birth, the following changes: the horizontal and vertical plates and the crista galli of the ethmoid were lacking, as well as the nasal bones. The upper jaw and the frontal bone were unnaturally fused together; only one rudimentary nostril was found, the septum was scarcely visible; there was fusion of the hemispheres and optic lobes; the corpus callosum, the third ventricle, and the pharynx were absent. The choanæ were closed by the palate, and numerous other anomalies were present.

Hill has described a tumor attached to the basi-sphenoid of a child which contained various dermoid structures and an embryonic form of liver by which, as Widley says, it may be elevated from the rank of a teratoma to that of a parasite.[1] Abraham has described a tumor containing dermoid structures and nodules of cartilage, covered with pilose skin, growing from the top of the pharynx near the basilar suture.[2]

In the museum of the Royal College of Surgeons is the enlarged head of a human fœtus with a large lobulated tumor, the remains of a second fœtus, growing from the median fissure of the palate: it projects into and dilates the mouth. This fœtus had a single nostril. The tumor contained fifty-nine vesicles, complex in form, closely crowded together. Accounts of other such tumors will be found in a paper by Widley.[3]

Arnold met with a congenital lipoma of the tongue and pharynx which perforated the sphenoid so that a portion as large as a walnut lay inside the cranium. The hard and soft palates were both cleft. The tumor presented hairs and sebaceous glands.[4] In a more recent paper he has collected all the cases which he could discover of pilose tumors growing in the pharynx. These cases are tabulated: in twelve of them cleft palate existed, and in two a bifid tongue.[5]

Samelson exhibited in Manchester in 1872 a patient twenty-three years of age with congenital defect of the frontal bone. This was covered by thickened integument, underneath which cerebral pulsation was visible. This is of the same character as failure to unite noted in cases of meningocele. A case similar to those spoken of by Hill and Abraham is described in the Pathological Transactions for 1867, page 251, by Lichtenberg. The tumor projected from the mouth, and its pedicle was found to perforate the sella turcica and to be connected there with an intracranial tumor.

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[1] Journal of Anatomy and Physiology, xix. 190.

[2] Ibid., xv. 244.

[3] Ibid., xxii. 428.

[4] Virchow's Archiv, liv. 82.

[5] Ibid., cxi. 176.

In the *Australian Medical Gazette* for March, 1888, Dr. Lendon has reported a remarkable example of an infant with two mouths. When the child was asleep it appeared as if it had only one mouth much larger than usual, and as though it were holding in this mouth a large fleshy mass, too large for it to swallow. This fleshy mass was attached for about one inch to the edge of the upper lip, to the left of the median line, and was evidently formed by fusion of the two adjacent cheeks, the line of fusion being marked by a faint median groove. It was attached backward along the roof of the mouth, and merged into what appeared to be a central pillar of the fauces formed by junction of the contiguous faucial arches ; around this central pillar a bent probe could be passed, showing that both mouths opened into a common pharyngeal cavity. By manipulation of this central mass the two separate mouths could be demonstrated very distinctly, and it was then seen that the right was much larger in every respect. There were faint indications of separate chins and lower lips. In the right mouth the uvula was normal, though displaced to the right ; in the left mouth the palate was cleft. The alveolar process of both jaws in both mouths could be completely traced. The right tongue was visible in its mouth, and the left tongue in its mouth, though the latter was much the smaller. A third or central nostril was seen, but it was of very small dimensions. The infant lived three months, and died of inanition.

As remarked above, failure to unite between the external part of the frontal process and the superior maxillary process is made evident by one form of oblique fissures of the face. This subject has been ably discussed by Morian.[1] He groups these fissures in three divisions, following the natural order as we find them occurring, as follows : *First*, the fissure begins in the soft parts laterally to the median line, as a typical hare-lip, penetrates into the nose, runs then beneath the ala, and, passing between the nose and the cheek, attains the lower lid or passes to the external angle of the eye, and frequently extends as far as the forehead. The bony defect begins at that point where the alveolar process is wanting in ordinary cases of hare-lip,—that is, between the intermaxillary bone and the intermaxilla, according to the older view ; according to the later view, between the internal intermaxillary on the one side and the external intermaxillary and superior maxilla on the other side. He refers to nineteen cases in this connection. In the *second* group the fissure which begins as a hare-lip does not pass into the nostrils, but runs externally to the nose, through the cheek, and up to the external canthus and the forehead. In the bony tissue the fissure is found between the external intermaxillary and the superior maxilla, and in more aggravated cases extends upward to the infra-orbital foramen. Twelve cases of this character are adduced, in the milder of which the nasal duct was intact, in the more severe was involved. In a *third* group, much more rare, the fissure begins in the soft parts at the external angle of the mouth and extends from there into the cheek, towards the external canthus. The bony fissure extends from the canine tooth towards the infra-orbital canal in the orbit. The palate remains intact; the tear-duct is closed. Only four of these cases are on record. These cases are most allied to those in which the defect extends from the mouth to the ear, with fissure of the cheek. Thus it will be seen that these fissures of the face may be unilateral or bilateral, complete or incomplete, simple or complicated. Of thirty-four cases which the author has studied, twenty-five were still-born; while ninteen were bilateral, nine had the fissure on the left side and six on the right side. He claims that numerous such cases undergo an intra-uterine closure. In five cases the fissures were combined with ordinary hare-lip, and in four cases with other defects of the cheek upon either side. In twenty-six of these cases other congenital defects of the skull or brain were noticed, such as hydrocephalus, encephalocele, etc. As far as the typical oblique forms are concerned, he considers that the first corresponds to the embryonal orbito-nasal border; that the second is due to a separation of the superior maxillary process from the external intermaxillary and to an excessive growth of the internal intermaxillary; and that the third is due to the separation of the inner portion from the superior maxillary process, as a result of amniotic adhesions.

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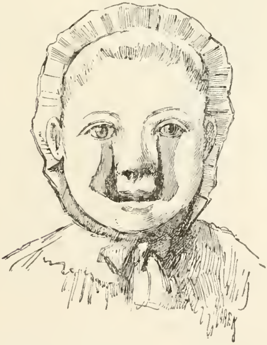
[1] Archiv f. Klin. Chir., xxxv. 245.

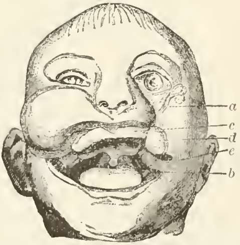
A remarkable instance of bilateral cleft between the nose and cheeks has been reported by Guersant.

FIG. 5.



Bilateral nasal cleft (Guersant's case).

FIG. 6.



HEAD OF A CHILD THREE DAYS OLD. Specimen in the Bonn Pathological Institute Museum. Described by Remacly. (From Trendelenburg.)—*a*, cicatrized fissure of face; *b*, enlarged buccal aperture; *c*, intermaxillary bone; *d*, alveolar process; *e*, a bridge of integument from the angle of the mouth to the gum.

Delpech described a patient who had congenital absence of the right nasal and lachrymal bones, of the nasal process of the superior maxilla, and of the soft parts.

One of the most remarkable specimens of complex defect is that described by Remacly and shown in Fig. 6.

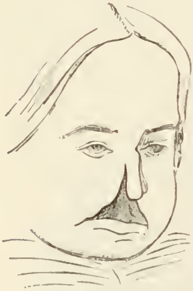
Thomas saw in a boy three months old a triangular opening on the right side of the nose, whose lower border corresponded to the opening of the nostril and whose upper angle was nearer the margin of the orbit. A small bridge of skin separated this fissure from the natural one between the eyelids.

Langenbeck has also reported analogous cases in which the nostrils were not closed, and in which the middle, external, and frontal processes had failed to unite.

Madelung has described and figured[1] a case of lateral fissure of the nose, not excessive in degree, but of interest on embryological grounds.

In a case observed by Bitot there had been no attempt at fusion between the upper jaws; there was an entire absence of all the parts formed by their meeting. Occlusion of the choanæ has been mentioned in a number of these cases: this may occur, however, without any extensive defect, and may even be unilateral. As evidence of minor failures of union in the median line we may mention also congenital fissures of the nose. Rusch saw a fissure at the lower end of the nose, in the case of a child, which opened into the nasal cavity. Beeny has also met with a congenital fistula upon the bridge of the nose. Lateral fissures of the nose may be met with in connection with hare-lip.

Fig. 7.



Lateral fissure of the nose. (After Broca.)

Broca observed a fissure of this kind in an eight-year-old girl. There was almost complete absence of the upper lip, and a fissure opening into the right nostril extending upward to within one centimetre of the internal angle of the orbit; the palate was also fissured. (See Fig. 7.)

Lateral nasal fissures have also been noticed in connection with coloboma of the upper eyelids. Coloboma of the upper or lower lids is occasionally seen in cases of fissures of the lip or palate.

Corresponding to the above as well as to the mildest form of cleft palate we have congenital perforations of the palate, which may vary in size from the smallest perceptible up to such openings as would be entitled to the name of cleft. Cases have been observed, also, of cleft in the bony palate without corresponding absence of mucous membrane, the bony defect being provided with a membranous obturator. These are found usually in the middle line and anteriorly.

DEFECTS OF THE NOSE.

Congenital absence of the nose is excessively rare. A number of cases are mentioned by Vrolik, who also speaks of one in which only the right half of the nose was present. Otto records a case of absence of the septum and consequently of one nostril. Cases are known also of a bifid condition of the extremity of the nose, the depression between the lateral cartilages having failed to fill up.

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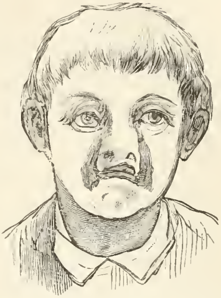
[1] Archiv f. Klin. Chir., xxxvii. 275.

Congenital atrophy of the nose is also known, and it frequently coincides with other atrophic defects of the face. On the other hand, a double nose has at different times been met with, one of the most remarkable instances of this kind being reported by Broleli in the case of a carpenter. Danyau has described a young woman provided with a double nose, one corresponding to each cheek. This patient also had three eyes and two rows of teeth. There are likewise cases of excessive overgrowth of this part.

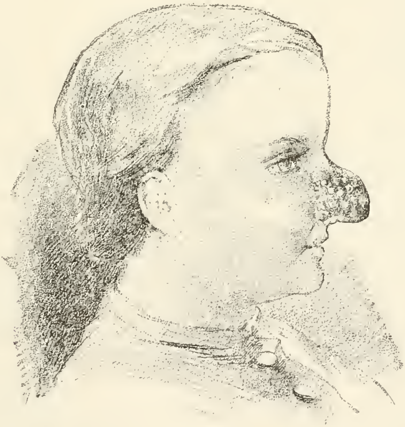
Fig. 8.



Double fissure separating the nose from the face. (Mason.)

Mothers' marks, or vascular tumors, are frequently found on the integument of the nose.

Fig. 9.



Vascular tumor of the nose. (Churchill.)

A case of what appeared to be three nostrils was seen recently by myself in a little infant. Apparently the three nostrils were distinct, and the external openings were of about the same size. The extra nostril was, however, merely a deep cleft in the septum of the nose, and extended to the depth of a centimetre and a half, where it terminated blindly. It was a simple matter to excise one of the walls of the depression and thus make this nostril communicate with the smaller of the natural channels.

In the *New York Medical Journal*, November 12, 1887, page 536, are reported two cases of congenital atresia of the nostrils, such as has also been observed by various previous reporters. This closure is usually membranous.

In the *Philadelphia Medical News*, November 10, 1888, will be found a valuable paper by Dr. Knight, dealing with congenital bony occlusion of the posterior nares. Posterior bony occlusion is much more common than bony anterior closure.[1]

DEFECTS OF THE MOUTH.

Atresia oris is sometimes a solitary defect, but more commonly it is combined with other malformations. It is of an adhesive or membranous character, the denseness or toughness of the occlusion varying in different degrees. In every case, however, some mark resembling a dimple will be found, denoting the point at which the closure has occurred, and serving as a guide to the surgeon. Simple incision, with care to prevent renewal of adhesion, is all that is required.

Microstoma is nearly always a milder form of the same occlusive process. The opening may be so small as entirely to prevent nursing. It will then call for immediate relief.

Macrostoma, on the other hand, is to be viewed as a defect or failure to unite, and belongs to the last class of fissures of the face mentioned above, provided it exceed the somewhat wide limits of relative size of the normal human mouth. It is, of course, to be remedied by a simple plastic operation.

The synechiæ of the mouth are divided by Jahr, following Gunther, as follows: 1. Synechiæ of the cheeks and gums, including (*a*) those of the angle of the mouth and lips with the gum; (*b*) those with destruction or defect of the lips; (*c*) those without participation of the lips: these may be direct or indirect, the latter being effected by fibrous bands or pseudomembranes. 2. Synechiæ of the tongue, including (*a*) synechiæ of the lingual apex,—in other words, a too short frænum; (*b*) synechiæ of the base of the tongue and floor of the mouth or oral cavity, these being direct or indirect, as by bands or tumors beneath the tongue; (*c*) between the margins of the tongue and gums; and (*d*) between the root of the tongue and the epiglottis. 3. Synechiæ of the posterior oral outlet, including (*a*) adhesions between the velum and the palatal arch, and (*b*) those between the soft palate and the posterior pharyngeal walls.

Such adhesions often accompany atresia of the mouth and call for a combined operation. Separation is usually not difficult, but care is required to prevent the formation of new attachments. When operating upon any such case, one should take pains to see that the entire upper end of the digestive and the respiratory tubes is made clear, providing the condition of the patient permit it. Adhesions of the tongue to the gums frequently accompany those of the lips to the gums, by which deglutition is very seriously interfered with and even expectoration and respiration seriously impaired.

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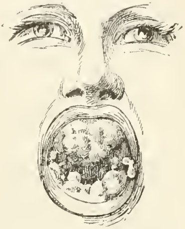
[1] See also an exhaustive paper by Hopmann, Archiv f. Klin. Chir., xxxvii. 235.

Congenital union of the gums is a deformity of exceeding rarity: it has been described by Littré and Bianchi in subjects presenting other irregularities, and has been observed also by Oberteuffer. As stated by St.-Hilaire, in Littré's case the closure of the gums was complicated with closure of the nares, the skin passing over both apertures. Dr. Carter has reported a remarkable case[1] of a male child born at term, which made spasmodic respiratory efforts. The finger was passed into the mouth, and complete union of the gums was found. Closure was effected by a tough membrane, one-eighth of an inch in thickness, passing from the palate-bone above and inserted along the gum of the lower jaw. Simple and complete division effected the desired result.

Congenital hypertrophy of the gums has been noticed by Salter, Gross, Heath, and others. Its progress has usually been rapid; the swelling may be symmetrical and may attain such a volume as to prevent closure of the mouth. A proper treatment consists in excision and cauterization of the exuberant growth.

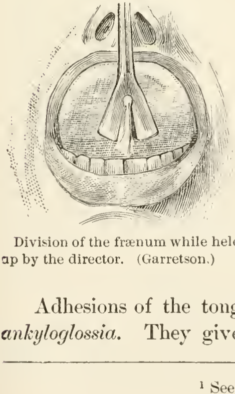
Fig. 10.



Congenital hypertrophy of the gums. (Follin et Duplay.)

Tongue-tie is the common name for a condition which may be classed as the mildest form of synechiæ belonging to division *b* of Jahr's second class given above. It is in effect a shortening of the natural frænum linguæ, by which projection of the tongue beyond the teeth is prevented. It is an impediment to the natural uses of the tongue, and interferes especially with perfect articulation. It varies in degree within considerable limits. Sometimes new-born infants are prevented by this condition from sucking properly at the breast, and their efforts are accompanied, as Dr. Dewees has pointed out, by a clucking sound.

Fig. 11.



Division of the frænum while held up by the director. (Garretson.)

No hesitation need be felt at any time about dividing the abbreviated frænum, save possibly in bleeders. Fig. 11, from Garretson, shows how the handle of the common grooved director is intended to be utilized as an aid in this little operation.

Adhesions of the tongue to the floor of the mouth have been termed *ankyloglossia.* They give rise to trouble in deglutition, mastication, and phonation, save in those cases in which the tongue is connected to the palatine vault, which would constitute *ankyloglossia superior*. In such cases deglutition and the other functions would be nearly or quite impossible: they are, however, extremely rare.

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[1] See Garretson's Oral Surgery, p. 433.

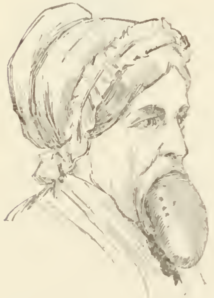
DEFECTS AND ABNORMALITIES OF THE TONGUE.

Total absence of the tongue has been noted in a very few and rare instances. Ambrose Paré described a most interesting case of a young person absolutely tongueless, who found that he could articulate better after introducing into the mouth a piece of wood shaped like the edge of a dish. Jussieu in 1718 saw a fifteen-year-old girl in Lisbon who presented an instance of this kind.

Arrest of development leads to the *microglossia* of various writers. This may be seen in all degrees. It is to be explained by simple failure to develop.

On the other hand, the opposite condition of *macroglossia* is more common. Most of the so-called cases of macroglossia are due to changes connected with the lymphatics of the organ; in other words, many of them are in fact lymphangeiomata of the tongue. In a few others, however, actual hypertrophy of muscular fibres has been made out, along with thickening of the vessel and nerve-sheaths. Degeneration has also been known to cause congenital enlargement, but this is probably also connected with lymphatic changes. The growth is said to reach an almost incredible size in some of these cases. Delpech has reported a case in which the organ attained ten times its normal size. Treatment in these cases must be surgical, and consists usually in excision or amputation. I have myself observed a case of extraordinary papillary growth of congenital origin, the tongue proper being scarcely enlarged, and yet, on account of the development of papillomata upon its sides and upper surface, it attained so large a size as to prevent closure of the mouth. A few months previous to my seeing the child, excision of the apex of the tongue had been made by taking out a wedge-shaped piece: the trouble continued, however, and I removed the anterior half of the tongue, at the same time cauterizing the bases of the other papillomata with the actual cautery. The child recovered promptly, and was soon able to breathe, eat, and sleep about as it should: subsequent tendency to papillary development was prevented by an occasional application of chromic acid.

Fig. 12.



Macroglossia. (Follin et Duplay.)

Lipomata or other congenital tumors developing in the tongue may cause genuine forms of macroglossia, while congenital cysts developing beneath it, either retention-cysts or those of new formation, may push it forward or distort it, and thus create spurious forms of the same trouble. Blood-vascular tumors in or beneath it may also have the same effect.

Bifid Tongue.—Another condition of considerable embryological interest, and one which reminds us of the forked condition of the tongue in many of the reptiles, is that of median cleft. This may be so slight as to be barely noticeable, or it may be quite extensive. A bifid condition of the tip of the tongue frequently coincides with division of the inferior maxilla and arrest of development of the lower part of the face, though it may be noted alone.

Butlin says that in cases of bifid tongue where the cleft is very long it is often associated with development of a tumor in the floor of the mouth. Ahlfeld has observed cases of cleft tongue coexisting with deep clefts of the face, and others have noted like phenomena.

At a meeting of the New York Pathological Society, Dr. Brothers presented a specimen of cleft tongue in a babe of one month. Cleft in the palate had been noticed at birth ; this had interfered with feeding, and there was consequent malnutrition ; the child had steadily pined away, and was dying of marasmus when seen by him. There was fissure of the soft palate extending up to the hard, and a cleft one-eighth of an inch deep at the tip of the tongue.[1]

Elongation of the tongue is another possible congenital change, and cases are on record where the tongue has been not so much hypertrophied as elongated, so as to be inconvenient of retention in the mouth. More rarely individuals seem to be born with the power of unusual motility of the tongue, so much so that the act of "tongue-swallowing" thus permitted is now known to be not so very infrequent.

PAPILLOMATA AND NEOPLASMS OF THE MOUTH.

These are conditions which may be either of congenital or of acquired origin. The former, which here concern us, are not so very rare. I have had the opportunity of examining the mouth of a young infant the mucous membrane of whose mouth and lips was studded with innumerable small warty growths that gave under the finger a sensation of velvet as they came in contact with it. Inside the gums there were none of these growths. The child apparently suffered no inconvenience, and I could only suggest the cauterization of various areas in succession, with internal administration of magnesia. The parents stated that the mouth of the child was in this condition when it was born.

Next to papilloma the angeioma is by far the most common. These are usually conveniently treated by electrolysis.

DEFECTS AND DEFORMITIES OF THE LIPS.

These are for the most part included under the headings *microcheilia* and *macrocheilia*. The former is inseparable from the condition of microstoma, of which it is an attendant feature. It amounts simply to a failure to keep pace in development with the surrounding parts. If by it the oral embrasure is made too small, a simple surgical procedure will afford relief.

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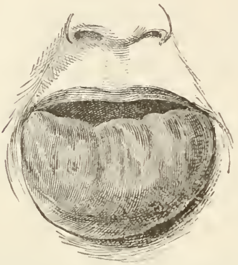
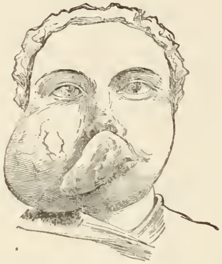
[1] New York Medical Record, January 28, 1888, p. 109.

FIG. 13.



Congenital erectile tumor of the lower lip. (Nélaton.)

FIG. 14.



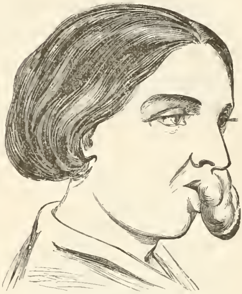
Congenital erectile tumor of the upper lip and the cheek. (Nélaton.)

Macrocheilia proper must be distinguished from deformities of the mouth caused by sloughing, malignant tumors, myoma, fibroma, etc., all of which frequently accompany deformities of the lips. Jacobi has described a connective-tissue form which is sometimes congenital, although Vidal has observed two similar cases which were brought about simply through repeated attacks of erysipelas. It is worth while to remember, as insisted on by Bardeleben, that through repetitions of erysipelatous inflammation permanent enlargement of the lips may be caused. Vidal has called this a *leontiasis*, pursuing an analogy to elephantiasis, and as such Volkmann has described it. These cases of macrocheilia through new formation of connective tissue must be largely ascribed to the *rôle* played by the lymphatics. They are usually classified as lymphangiectatic microcheilia. Billroth, Langenbeck, and Wegener have described or operated upon numerous cases of great interest. In the latter's case the malformation was connected with an overgrowth of the upper jaw. Dolbeau and Felizet have described a very rare combination of cavernous lymphangioma and fibroma. Their case was an infant of nine months with an enormously overgrown upper lip, of congenital origin. A large portion of it was removed by plastic operation; excessive hemorrhage occurred, but recovery from the operation followed, though the child died a little later of tuberculous meningitis. Another form of macrocheilia is constituted by diffuse angeioma, usually of the upper lip. In these cases the trouble may be confined to the lip alone, or, as in one which came under my notice, may involve both lips and the entire thickness of the cheek, the muscular tissue having been principally absorbed away.

This same form of venous tumor may occur in the inside of the mouth. I have recently had under treatment, and cured, a lady who had a tumor of this kind in the inside of the mouth, spreading on the ramus of the jaw and down to the fauces. The remedy in her case was electrolysis. In another case, where the lower lip, upper lip, and cheek were covered with such a tumor, I resorted to a combination of electrolysis and ligation.

Bouisson collected ten cases of macrocheilia, the nature of the trouble being vascular tumor, and found that six times it occurred on the lower lip, twice on the upper lip, once at the commissure, and once it extended entirely around the mouth. These vascular tumors are in no wise different from those met with in other parts of the body, their character being either that of the venous angeioma or that of the cavernous tumor. They are to be dealt with here on the same general principles as elsewhere,—by electrolysis, by ligature, or by excision, according to the taste of the operator and the nature of the case.

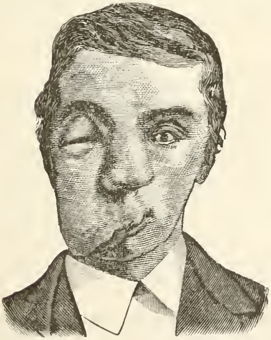
FIG. 15.



Venous angeioma. (Garretson.)

Under the name *dermatolysis* Garretson has described an hypertrophy of the integuments or of the soft parts much resembling the deformity caused by elephantiasis. The surface is rugous and more or less pigmented. It has been also described by Hebra and Kaposi as of purely congenital origin; is spoken of by Duhring as a variety of molluscum fibrosum; and is stated by Cooke not to make its appearance after puberty. This may attack face or lips, as shown in Fig. 16, from Garretson.

FIG. 16.



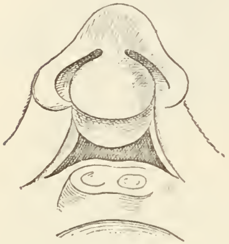
Dermatolysis. (Garretson.)

Other defects of the lower part of the face are produced by irregularity of development, inducing a deformity which may be slight in degree, as seen in many individuals, or may be a prominent and unsightly deformity. Congenital smallness of the lower jaw may be combined with fissures or with irregular development of the ear, or may be met with by itself.

Parise described the case of an infant fourteen days old, with complete median fissure of the lower lip and division of the lower jaw into two portions, which remained three millimetres apart and were joined by connective tissue. There was also fissure of the tongue, which extended quite a distance backward in the floor of the mouth. A defect was found between the upper borders of the genio-glossi muscles. The apex of each half of the divided tongue was found bound down by too short a frænum. Most of these fissures of the lip are median, but Fergusson has reported one case of lateral fissure close to the left angle of the mouth extending to the lower border of the jaw.

One of the most curious of congenital defects is that of *fistulæ of the lower lip,* which were first noticed and described by Demarquay at a point close to the middle line. In a case reported by Rose were seen two symmetrical fossæ, which led down into fistulæ whose passages terminated by a blind extremity about three centimetres from their opening. The two fistulæ ran in the thickness of the lip somewhat nearer the mucous membrane than the skin. In this case there were other congenital defects, including a well-marked fissure of the upper lip. The nature of these fistulæ, which have been noted but a few times, is very hard to explain: they are lined with membrane, although it is not generally known that they secrete any fluid. By themselves they constitute a defect of slight importance, but they are of extreme interest from an embryological point of view. Rose's case is illustrated in Fig. 17, which also shows deformity of the upper lip.

Fig. 17.



Fistulæ of the lower lip. (Trendelenburg.)

Fritsche has reported an extraordinary instance of a somewhat similar defect in the case of a female infant of five months. He found an opening on the margin of the lower lip; from this a sinus ran downward and inward in the thickness of the lip and cheek and terminated underneath the mucous membrane, which appeared to be lined by mucous membrane, and which when the child cried appeared to open and discharge some drops of fluid. A communication with the mouth could not be found.[1]

DEFECTS OF THE LOWER JAW.

There are cases on record in which the lower jaw does not match the upper jaw at all. These are usually accompaniments of fissures of the cheek and lips, with other abnormalities. Langenbeck reported a case in which he could pass his hand into the child's mouth and feel that the ascending ramus was entirely absent, and then he could pass his finger up in the proper direction and feel the glenoid cavity of the temporal bone and assure himself that it was empty. Deficient lower jaws usually have too few teeth.

The most conspicuous forms of deformity of the lower jaw are comprised under the terms *epignathy* and *polygnathy.* Instances of epignathy have been most commonly met with in connection with fissures of the lower lip and lower jaw, and in the direction of tumors or hypertrophic forms of connective tissue. Polygnathy, however, is constituted by peculiar or unusual embryological development, in which case we have to consider the rudimentary second jaw either as the remnant of a second individual or else in the light of a teratological tumor. Thus, Meyer has described one case, met with in the clinic at Bonn, in which the second rudimentary lower jaw contained nearly a complete maxillary arch, with incisor and molar teeth planted upon the outer surface of the lower half of the unnatural jaw, the whole constituting a tumor which was removed by operation. He also refers to another case in which only half of the supernumerary lower jaw was found. A discussion of such cases here would lead us too deeply into the mysteries of teratology, and those interested are referred especially to the encyclopædic work of Geoffroy St.-Hilaire, and to the article "Teratoma" in the "Encyclopædia of Anatomy and Physiology."

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[1] See also Archiv f. Klin. Chir., xxxvii. 271.

DISEASES OF THE MOUTH.

BY W. H. ALLCHIN, M.B., F.R.C.P.

AFFECTIONS of the mouth are of very common occurrence in infancy and childhood, and various circumstances, both intrinsic and extrinsic, separately or combined, tend to favor this frequency. The exposure of the buccal cavity to the admission of all forms of irritants is obvious, and a natural habit of young children to thrust anything and everything into their mouths is well known. Consequently, the risk of injury from the entrance of sharp or hard substances, of materials which are acrid or caustic, too hot or unduly cold, has always to be considered; whilst the admission and lodgement of germs and spores of fungi are facilitated by the conditions of artificial feeding and the greater difficulty of keeping the cavity clean in the very young. During the early period of life, also, there normally occur changes in the mouth associated with the eruption of the teeth which of themselves confer a very distinct liability to the occurrence of disease, and this notwithstanding the fact that dentition is accomplished in many cases without discomfort or ailment. Whether or not the greater vitality of the tissues in childhood renders them more susceptible to disease cannot be affirmed, but it would certainly seem that their reparative power is then at its maximum and so tends to characterize the course that the maladies follow.

It is usual to consider that affections of the mouth are very frequently due to extension from or sympathy with diseases lower down in the alimentary tract. This idea has come to be accepted almost without question, but it is not easy to see the grounds for such a belief, and there is much in the writer's view that is opposed to its general truth; for, although, no doubt, a gastro-intestinal disturbance may be accompanied either as sequence or coincidence with a stomatitis, it is certain that a large number of cases will offer no such complication, and an equally large number of mouth-troubles may arise and run their course without any appreciable or recognizable alteration in the functions of the stomach or intestines. In the writer's judgment, it is a mistake to ascribe, as is so commonly done, the greater number of inflammatory conditions of the mouth to an existing gastro-enteric perversion. And although bad feeding and improper hygienic surroundings are responsible for many affections of the mouth as well as of the stomach and intestines, it is not by any previous dyspepsia that the former are caused, but rather they are as much primary in origin as are the latter.

Diseased conditions of the mouth are conveniently divisible into those which are primarily or mainly limited to that region and those in which the altered state of the mouth is but a part of some more general affection. These last will receive here but little more than mention, and reference for further information must be made to the appropriate sections.

The special diseases of the mouth are almost entirely inflammatory in character, and, with one notable exception, are confined to the mucous membrane. All degrees of inflammation are met with, from the mildest erythema and catarrh to extensive ulceration or even gangrene. Owing partly to the various theories that have been held as to the pathology of inflammation, and partly also to the differences that undoubtedly do exist between what may be called typical cases, there has been a very extensive and very variable subdivision of stomatitis into groups, with an excessive and not always consistent nomenclature, which have all tended to confusion. The arrangement adopted in the following sections is that which appears to the writer the natural one; but it must be remembered that no very sharp lines exist between the varieties described, and that there are cases of frequent occurrence which occupy a connecting position, thus going far to support the view that they are but stages of the inflammatory process, differing mainly in their severity as determined by the nature of the cause or the constitution of the patient, or both. Sufficient differences, however, do exist clinically to justify a separate description, which will be pursued under the following heads:

Simple or catarrhal stomatitis.
Parasitic stomatitis.
Stomatitis accompanied with ulceration.
Gangrene of the mouth.

No practical benefit would result from any attempt at a detailed historical sketch of these maladies. Several of the names now in use, such as aphthæ and noma, date from the time of Hippocrates, but certainly not with the same signification they now possess; and, since the diseases of the mouth as we now regard them are for the most part varieties of inflammation, no rational subdivision could be made until this pathological process had itself received a description and definition. Much difficulty also is met with in following the accounts of these affections by the older writers, from the variety of meaning attached to the names employed, a source of confusion which even some recent authors have not avoided.

SIMPLE OR CATARRHAL STOMATITIS.

Synonyme.—Stomatitis erythematosa.

Definition.—These terms denote a moderate inflammation of the buccal mucous membrane, which is usually acute in its course, unattended with danger, and does not proceed to ulceration.

Etiology.—The disease occurs both as a primary affection and as secondary to remote or general morbid states. The former are the more frequent, and are those more especially calling for notice. Among the causes directly connected with the mouth, and therefore to be regarded as primary, is dentition, which is a normal physiological process especially prone to set up inflammation to a moderate degree, although its occurrence is by no means invariable. The condition is, therefore, commonest within the first two years of life, and the greater number of cases perhaps are met with during the first twelve months. There does not seem to be any greater liability to the occurrence of the malady in the ill-nourished or cachectic; it frequently develops in the perfectly healthy. The habit, that infants and young children so commonly have, of carrying everything they can lift to their mouths makes mechanical irritation an occasional cause of stomatitis, and in this connection prolonged sucking at an artificial teat or an imperfectly-developed nipple may be mentioned. Another and not infrequent group of causes are ingesta of an irritating character, such as too hot or too cold food, or too highly seasoned or very acid articles of diet. An excess of sugar would seem in some cases to induce the condition. And, lastly, exposure to cold, either by draughts of cold air or by the child getting wet feet or "wet through," may produce a catarrh of the mucous membrane of the mouth, as it may that of the respiratory tract or of the lower portions of the alimentary canal.

Catarrhal stomatitis may be secondary to gastro-intestinal derangements, especially those of an inflammatory character. It may be associated with the various ulcerative processes that affect the tonsils and pharynx, and is commonly met with to a varying degree with the different forms of ulceration of the mouth to be presently described. Of more interest, although not of practical importance, is the simple stomatitis which frequently accompanies some of the acute specific fevers, especially measles and scarlet fever.

The condition very rarely becomes chronic in children, although it may be more or less continuous through the entire period of dentition.

Morbid Anatomy.—The structural changes in the mucous membrane constituting the disease are those of an ordinary catarrhal inflammation. There is a marked increase of redness, which is usually of a bright tint, though sometimes livid, over the inside of the cheeks and lips, and to a somewhat less extent where the mucous membrane is thinner and more adherent to subjacent structures, as on the gums and hard palate; indeed, in the latter situation and along the line of eruption of the teeth, should they not be cut, the surface may even be pallid. The hyperæmia may be almost or quite uniform, or it may exist in patches causing a mottled appearance, which is particularly the case in the stomatitis accompanying measles. The redness is very apt to become less marked after the disease is well established, owing to the proliferation of the epithelium, which takes place especially on the inside of the cheeks and on the filiform papillæ of the tongue, where it contributes with various organisms and débris of food to form the fur; patches of the thickened epithelium may also be seen on the gums. The increased vascularity with the epithelial overgrowth and a small amount of effusion from the vessels into the tissues determine a pearly-white swelling of the mucous membrane, although the extent to which this takes place is never great, being chiefly apparent on the inner surface of the cheeks and tongue, which are liable to be indented by the teeth if they be through the gums. The inflammatory process further shows itself in an increase of the buccal fluids,—saliva and mucus,—which are at first thick and viscid, but later slimy and watery, occasionally of an acid smell, but never offensive. The submaxillary lymphatic glands are rarely, if ever, enlarged, unless the affection have proceeded to ulceration; but the mucous follicles are frequently swollen and prominent, the obstruction in the ducts sometimes leading to the formation of cysts.

Although the inflammatory process usually involves the entire mucous membrane of the oral cavity, it sometimes occurs that the tongue or the gums alone are affected. A simple gingivitis is the more frequent: glossitis will receive separate description.

Under the heading Simple or Catarrhal Stomatitis are included the mildest cases in which the destructive phase of the inflammatory process does not amount to the actual loss of tissue or to ulceration. The limitation is necessarily somewhat arbitrary and artificial, since a very slight extension of the inflammation will lead to a superficial ulcer, and no absolute distinction can or should be made between the cases which just stop short of such a result and those in which it occurs.

It is a fact, though the explanation is not obvious, that the mucous membrane of the mouth and, indeed, of the entire alimentary canal is far more frequently the seat of catarrh in children than the respiratory mucous membrane. Between these two membranes there is a further difference as regards the inflammatory product, for, whilst from the air-passages there is very soon thrown off a muco-purulent or purulent discharge, that from the mouth rarely, if ever, at least in children, presents such characters, and amounts to little more than an excess of the normal buccal secretions,—rather more diffluent perhaps, and slightly turbid from desquamated epithelial cells and few leucocytes, but the latter are not found in such abundance as in a catarrh of the nasal or bronchial mucous membrane.

The process of recovery, which may take place in a very few days, consists in a subsidence of the hyperæmia and arrest of the epithelial proliferation; the surface frequently presents for a short time a denuded appearance or even superficial erosions, but no actual destruction of the mucous membrane or ulceration. A papillomatous induration of the membrane has been noticed as following on very prolonged cases.

Symptoms.—The objective signs of the affection are comprised in the account of its morbid anatomy. The febrile state, as is so frequently the case in children, is of great variability, and affords little or no indication of diagnostic or prognostic value; in the majority of cases the temperature is not more than a degree or a degree and a half above normal; occasionally it may rise to 103° or 104° F. The inflamed state of the whole surface of the oral cavity is accompanied with a sense of discomfort, varying from a troublesome itching to positive pain and soreness, though rarely sufficient to prevent adequate feeding. The salivation already alluded to is commonly preceded by a stage of heat and dryness in the mouth, often appreciable by the mother when suckling, at which time the surface is at its brightest color. The altered condition of the secretions and of the epithelial covering is liable to cause perversions of taste, even amounting to a bitter or disagreeable flavor, though it is only by older children that such is complained of.

In the majority of cases there are no further symptoms, but sometimes signs of gastro-intestinal disturbance manifest themselves, such as refusal of food, diarrhœa, flatulence, and pain in the belly. The relation of such a condition to the stomatitis is uncertain; both may be due to a common cause, or there may be reason to think that the dyspeptic trouble is secondary to the mouth-state, or the reverse may be the case. The easily-disturbed reflex centres of children are liable to respond to the peripheral irritation caused by the local inflammation, and all degrees of restlessness, fretfulness, irritability, and sleeplessness may be met with, which are more fully considered under disorders of dentition.

Diagnosis.—The recognition of the disease is not difficult. Very frequently attention is drawn to this condition in infants by the increased salivation and by the disinclination they exhibit to thrusting their fists or other objects into their mouths, or by the evidences of pain that ensue on taking food, whether by spoon or by nipple. Such circumstances should suggest an examination of the mouth, when the appearance of the mucous membrane will reveal the disease.

Prognosis.—Catarrhal stomatitis is not of itself a serious affection, and is never fatal. As a feature of gastro-intestinal disorders or of the exanthemata it may share in the gravity which such maladies confer. Its duration is variable, as the cause may persist or be removed, but its natural tendency is to recovery when the mucous membrane can be placed in such condition that the recuperative power may assert itself, and it leaves no subsequent evidences.

Treatment.—Very many cases recover without any special treatment when the cause has been removed or has ceased to operate. The catarrhal inflammation associated with a prolonged and troublesome teething may require direct attention. It is very desirable that the state of the bowels be regulated, and a laxative, such as castor oil or confection of senna, in doses according to age, is frequently very beneficial. Should there be any diarrhœa it should not be checked by astringents, unless, which is unlikely, it become profuse, and then a dose of chalk-mixture is to be preferred.

In consequence of the pain and soreness in the mouth, some tact is often required to give the necessary food. This should be entirely fluid: milk alone is usually sufficient, and is less irritating when cold. To wean a young infant on account of simple stomatitis would not be desirable, but it may require some perseverance to make the child suckle.

Locally, the objects are to allay the soreness and subdue the inflammation. The former indication has been met by mucilaginous decoctions, such as of marshmallow (Althæa officinalis) or of linseed, a mouthful of which may frequently be taken before food and then spit out; or for infants they may be painted over the mucous membrane of the cheeks and lips with a camel's-hair brush. It is not usual, however, for the pain to require any such application unless there be ulceration. More useful are mouth-washes of chlorate of potassium or borax (fifteen grains of either to one pint of water). Older children may be taught to rinse their mouths out frequently with such a wash, and for infants it may be applied with a syringe or spray-apparatus, the child being suitably inclined forward to allow the escape of the fluid from the mouth.

In the rare cases in which simple stomatitis becomes chronic, local astringent applications may be required (*e.g.*, argent. nit., gr. iii, ad aq. ℥i), with tonics or change of air.

The restlessness and irritability are safely allayed and sleep induced by small doses of bromide of potassium (gr. ii to x, according to age), with syrup. rhœados, ♏xx, ad aq. ʒi.

PARASITIC STOMATITIS.

Definition.—An affection of the lining membrane of the mouth essentially characterized by the development of certain fungi; determined by a previous unhealthy state of the mucous surface, which is apt to become slightly inflamed in consequence.

Synonymes.—Thrush, White mouth, Sprue; French, Millet, Blanchet, Muguet; German, Soor; Swedish, Torsk; Dutch, Sprouw.

Etiology.—The specific organism producing this affection, saccharomyces albicans, is a member of the order saccharomycetæ or yeast-fungi, of the class of achlorophyllous thallophytes, and presents the following characteristics. Cells round, oval, or cylindrical,—the former .003 to .005 millimetre in diameter, the last of the same thickness, but ten to twenty times as long, forming mycelium-like filaments which give off branches at the constrictions between adjacent cells and "from which by lateral and terminal gemmation spring spherical or oval torula-cells. It also forms ascospores containing four to eight spores."[1] Both mycelial filaments and spores contain dark granules which may exhibit Brownian movement. "They can be easily cultivated in a nutrient solution containing sugar and ammonium tartrate. The cells germinate according to the richness of the fluid in sugar; they either grow into long threads, or in a very strongly saccharine solution many daughter-cells are formed, budding out in various directions."[1] Considerable polymorphism of the fungus is determined by the nature (solid, liquid, etc.) of the medium in which it is cultivated.

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[1] Klein, Micro-Organisms in Disease.

The fungus was first described by Berg, of Stockholm, in 1842, and in the same year by Gruby. It was named oïdium albicans by Robin (1853), and continued to be so called until its alliance with other members of the group saccharomyces had been shown. Hallier stated it to be the same as oïdium lactis, which causes the sourness in milk, and in 1886 Grawitz extended its identity to saccharomyces mycoderma or mycoderma vini; the complete resemblances, however, are open to doubt.[2]

A malady which consists essentially of the growth and development of an organism will, of course, be susceptible of ready propagation by contagion, and the disease only occurs when the spores gain entrance to the mouth. To what extent they are present in the atmosphere remains uncertain, and how far sour milk may be a vehicle of contagion must depend on the settlement of the identity of the oïdium lactis with the specific fungus of thrush; but there can be no doubt that in the greater number of cases it originates from spores carried by spoons, feeding-bottles, teats, etc., used in the artificial feeding of the infant. The mother's nipple may also become a means of conveyance from babe to babe. Hence it is that the malady may sometimes assume an almost endemic character in institutions where infants are nursed and tended.

A circumstance of great importance connected with the occurrence of the disease is that the growth and development of the fungus will not take place unless the mucous surface or secretions are abnormal. In a perfectly healthy mouth, though the organism may enter and even be found therein, it will not flourish. It is then as an evidence of disease rather than as a special disease itself that thrush should properly be regarded, and in strict pathological accuracy of expression the affection should not be spoken of as a form of stomatitis, since an inflammation of the mucous membrane is not an essential of the disease. Moreover, it may be asserted generally that the extent to which the fungus does develop is proportionate to the extent of the perverted nutrition of the mucous surface, of which, indeed, the amount of thrush may be taken as a rough index.

The altered condition of the mouth which would seem to be essential for the growth of sprue is an acid state of the secretions. Normally the oral mucus and saliva are alkaline in reaction, but, largely owing to the smaller portion of the latter fluid in infants, they very readily become acid, and are frequently found so for a short time after birth. The changes in the media induced by the very growth of the fungus result, among other things, in an acidity, and no doubt a large share of the acid state of the buccal secretions which is invariably found with thrush is due to the fungus itself, though some acidity to start with appears to be necessary. Acid fermentation of the remains of milk, sugar, etc., remaining in the mouth, may be sufficient for the purpose; and constant sucking of sweetmeats serves to encourage further growth by providing a suitable pabulum for the spores and by facilitating the acid fermentation. Some experiments on the cultivation of the saccharomyces albicans in different media by Audrey[1] went to show that growth will take place in fluids which are neutral, or even slightly alkaline, though becoming acid as development proceeds.

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[1] Crookshank, Bacteriology, 1887.

[2] The similarity of the fungus to those members of the ascomycetes (a group of the order hyphomycetæ or mould-fungi) known as tinea, trichophyton, etc., which give rise to special skin-affections, may be noticed. The difference between the orders saccharomycetæ and hyphomycetæ consists chiefly in the hyphæ or segments of the latter forming special organs of fructification or gonidia, whilst in the former multiplication takes place by simple budding. (See Plate, Figs. 4 and 5.)

Almost any alteration in the condition of the mucous membrane may be sufficient to afford a suitable nidus for the growth of the epiphyte, and thrush may quickly follow on the mildest catarrhal stomatitis or complicate the profound malnutrition of the tissues established by congenital syphilis or by severe prolonged gastro-enteritis. It is in this way that improper feeding and bad hygienic surroundings contribute to the occurrence of the disease, leading, as they do, to deteriorated health and thereby furnishing a mucous membrane with impaired vitality. Warm weather, by tending to digestive disorders, also predisposes to the development of the fungus.

A consideration of the conditions which favor the occurrence of thrush will explain why the affection is far commoner in the earliest weeks and months of life. Its appearance and continuance after the first few months is generally in association with some severe acute illness or some wasting disease, just as in later life it may appear in phthisis. Cases developing within the first few days after birth have been regarded as infected from the vaginal mucous membrane during parturition.

Before the characteristic patches of thrush are visible, spores of the fungus may be seen in the scrapings from the mucous surface. The patches themselves have a pearly-white appearance, are slightly raised above the surface, sometimes to the thickness of a line, and generally thicker in the centre; they are at first firmly adherent to the membrane, and consist of the spores and mycelium of the fungus which surround the epithelial cells, upon which they form a dense felt and between which they extend even down to the mucosa, together with occasional filaments of leptothrix, bacilli, granular débris, and fatty molecules. The spots vary in size from a pin's point upward, occasionally covering a considerable area. They may be but few or they may be extremely numerous. As they become older they lose their bright, clear appearance and turn brownish or yellowish, at the same time becoming looser and more easily rubbed off.

As regards their distribution in the mouth, their commonest situations are the dorsum and edges of the tongue, then the lips, cheeks, and hard palate, being sparser and less frequent on the gums and the soft palate. It is very unusual to find the fungus on the tonsils or pharynx without its being present also in the mouth, but exceptional cases are recorded, and one of a very instructive character by Dr. Tordens,[1] where the affection was restricted to the soft palate and pillars of the fauces in an artificially-fed infant six months old. The explanation of the case appears to lie in the circumstance that after feeding the child's mouth was always washed out with alkaline Vichy water, which neutralized any acidity in the mouth and so prevented the formation of thrush there, but that the fluid probably did not reach to the back of the mouth, where the organism could germinate without hinderance.

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[1] Rev. de Méd., Paris, 1887, p. 587.

The subjacent mucous membrane is smooth, of a bright or livid red, and in severe cases of marasmus, when the mycelium has penetrated deeply, the patch when removed may leave a small ulcerated bleeding surface; but in mild cases the growth when rubbed off leaves the surface intact and of smooth shining appearance due to loss of the epithelial cells.

Associated with the fungus may be a mild stomatitis that has prepared the soil, as it were, for the growth of the parasite, which in turn may occasion a further degree of inflammation of the surrounding mucous membrane; or the stomatitis may be more considerable, due in great measure to any coexistent gastro-intestinal disturbance. In other cases the membrane may appear quite healthy.[2]

Symptoms.—Symptoms directly attributable to the thrush can scarcely be said to occur. When the formation is very excessive it may offer some obstruction to swallowing, but such a condition is exceedingly rare, and usually there is but very slight discomfort in the mouth beyond a dryness, though in severe cases soreness sufficient to interfere with sucking may be present.

Many of the symptoms often described as belonging to thrush are really those of the coexisting stomatitis. When associated with wasting diseases the amount of the growth will be probably much more extensive, but any symptoms such as diarrhœa, emaciation, debility, coldness in the extremities, anæmia, ulcers on the malleoli or heels, etc., are referable to such diseases, and not to the thrush which has been encouraged by the state of malnutrition. A distinction, therefore, between mild and severe cases of thrush has no rational basis beyond indicating the general state of health of the child.

Since the affection is so commonly associated with gastro-intestinal catarrh and diarrhœa, it frequently occurs that, from want of proper cleanliness or from the acrid nature of the evacuations, the anus and adjacent skin become red, erythematous, and even excoriated. This is vulgarly regarded as an indication of the "thrush running through the patient," and, should improvement in the mouth-condition happen to coincide, is regarded as a favorable omen. The real grounds for such a belief are very shallow. For although the saccharomyces albicans has been met with in the œsophagus, where it may form an extensive coating, and far less often in the stomach, small intestines, and cæcum,[1] its occurrence is by no means certain, and it certainly is not the common cause of the eruption referred to, which is determined in the way mentioned, especially in cases of congenital syphilis, where the skin is unduly sensitive to irritation. Dr. Goodhart[2] is inclined to believe that the superficial dermatitis on the buttocks and genitals is due to excess of starchy food, causing a vitiated blood-state, since diminution of such food is often sufficient to cure the condition.

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[1] Jour. de Méd., Chir. et Pharm. de Bruxelles, 1885.

[2] Those who are specially interested in the subject of this article are referred to the series of papers on "Diseases of the Mouth (Non-Surgical)," which have appeared during the year (1889) in the Archives of Pediatrics by Dr. F. Forchheimer.—Ed.

Diagnosis.—Any doubt that may exist as to the nature of white patches seen on the mucous membrane of the mouth will be immediately settled by microscopic examination, but it should be remembered that small portions of the specific fungus of thrush are sometimes to be found in diphtheritic membranes.[3] The ease with which they may be removed from the surface will suffice to distinguish small spots of milk curd, to which thrush has a close resemblance; and the dryness of the mouth is in noticeable contrast to the salivation accompanying aphthæ.

Prognosis.—Of itself thrush is without danger, though it may be significant of a deteriorated state of health which has permitted its development, and as such it is undoubtedly of grave import. When appearing in otherwise healthy infants the patches last but a few days, but in cachectic children they may continue for months, fresh spots appearing and extending as others soften and break away.

Treatment.—As regards the prevention of the disease, it is practically impossible to avoid the entrance of spores into the mouth; all that may be hoped for is to keep the mucous membrane and secretions in a condition which will not afford them a soil for development. Since, however, stomatitis is so easily induced, this becomes very difficult of attainment, even apart from the occurrence of grave constitutional maladies which are beyond our present means of prevention except so far as they may be controlled by proper feeding and attention to general hygiene. With a little care, however, much may be done in prophylaxis by strictly keeping the mouth clean and by carefully washing it with dilute alkaline fluids, such as Vichy water or lime-water, after every meal, thus removing the remains of food and diminishing the chance of acidity.

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[1] The existence of thrush on the mucous surface beyond the limits of the squamous epithelial covering—*i.e.*, at the cardiac orifice of the stomach—was for a long time denied, but there is no doubt of its occasional appearance in the above-mentioned situations. It is strange, however, that it should never be seen on the mucous membrane of the respiratory tract, except very occasionally on the true vocal cords, where the epithelium is non-ciliated.

[2] Diseases of Children, 1886.

[3] It should be remembered that there is reason to regard certain micro-organisms as normally infesting the oral cavity. Vignal (Arch. de Physiol. Norm. et Path., Paris, 1886) has described nineteen such in the adult. Whether any of these can be regarded as also normal in children or in infants before the teeth are cut is unknown, but at least their occasional occurrence should not at once be regarded as of morbid import.

When the patches appear, it is desirable, if possible, to rub them off with the finger covered with a handkerchief; but this is not always easy, and would not of itself be sufficient, since some spores are certain to remain. It is rather to kill the growth that efforts must be made, meanwhile keeping the mouth alkaline. These indications are met by borax or by sulphite of sodium, which have been shown to be fatal to the development of the fungus. Various methods are adopted for their application; the most usual, perhaps, and in some respects the most effective, is to paint over the surface of the mucous membrane with a camel's-hair brush a solution of borax in some viscid fluid, thereby insuring its more prolonged contact with the patches of fungus. The excipient commonly employed is a mixture of glycerin and honey in the proportion of one part of the former to sixteen of the latter containing two parts of borax. The sweetness of such an application makes it palatable to children, but the sugar is, nevertheless, distinctly detrimental, as it supplies an excellent pabulum for the fungus: this objection is to a great extent met by using glycerin only. The writer, however, much prefers that the application should be made in the form of a spray. The nozzle of the apparatus is easily introduced into the mouth, and the jet of liquid can be effectively directed to every part. Solutions of borax or of sodium sulphite (one drachm of either to an ounce of water to which a little glycerin has been added) may be applied in this manner every hour or two, with most beneficial effect. The value of chlorate of potassium for thrush alone is but slight, though should there be much coexistent stomatitis ten or fifteen grains may advantageously be added to either of the above solutions. As a general rule, the sodium salts are to be preferred in this condition to those of potassium, and the bicarbonate only may be sufficient.

It should be scarcely necessary to add that the strictest attention must be paid to the diet, upon principles elsewhere laid down; but it is advisable to diminish the amount of sugar and starchy food that is given, and if the infant be only taking milk to add thereto a fourth part of lime-water. In addition to the proper food for the age of the child, it is frequently necessary to supplement the feeding with a very small quantity of alcohol (a few drops to a drachm, according to age), which is preferably given in the form of good brandy with the milk or water. The writer is fully convinced of the absolute necessity of this course in very many cases when the vitality of the child is much diminished, considering that the difference between death and recovery may often depend upon the prompt and sufficient administration of this drug. Port wine, sherry, or rectified spirit may be used in place of brandy, though scarcely with advantage. When the general health is much deteriorated, tonics may be requisite, and such formulæ as the following are recommended:

℞ Ammon. carb., gr. iss;
Tinct. cinchonæ comp., ♏x;
Tinct. cardam. comp., ♏v;
Aquæ, ʒi.

℞ Acid. nit. dil., ♏iii;
Tinct. cinchonæ comp., ♏x;
Glycerini, ♏xv;
Aquæ, ʒi.

In specially anæmic cases iron may be given in the form of vin. ferri amarum, or—

℞ Tinct. ferri perchloridi, ♏ii;
Potass. chlor., gr. iii;
Aquæ, ʒi.

or—

℞ Ferri et ammon. cit., gr. iii;
Glycerini, ♏xv;
Aquæ, ʒi.

These doses are suitable for infants two or three months old.

It is often needful to initiate the treatment with an aperient in the form of a rhubarb-and-soda powder, with or without hydrarg. cum creta, and to repeat the same every few days.

It is rare that any special treatment is required for the ulcers left by thrush in debilitated children. Reliance must be placed mainly on constitutional treatment, but weak solutions of nitrate of silver or of sulphate of copper may be applied with a brush: in such subjects the use of the solid crystals is objectionable, as the tissue-destruction is thereby extended without corresponding advantage.

As a valuable preventive measure, the feeding-bottle, tube, artificial teat, spoons, etc., should be kept scrupulously clean, and the glasses scalded daily and kept in a solution containing one drachm of salicylate or sulphite of sodium to the pint of water.

LA PERLÈCHE.

This affection was first described in 1886 by Dr. Lemaistre, of Limoges, who found it to be extensively prevalent among the children in the villages of Limousin in France. It has received its name from the smarting sensation in the lips causing the children constantly to lick (*perlécher*) them. The disease is confined to the angles of the mouth, where the epithelium becomes thickened, the superficial layers easily separating off, but rarely denuding the derma; sometimes there are small fissures, which are painful, especially on opening the mouth. The appearances closely resemble the mucous tubercles and fissures of congenital syphilis. It lasts from fifteen to twenty days, giving rise to no other symptoms. Microscopically the epithelial thickenings are always found to contain numerous sphærobacteria, singly or in chains or masses, infesting the edges of the epithelial cells, which may be invaded and destroyed. Careful examination discovered similar cocci in the water of the neighborhood, which were shown to be directly conveyed to the children and by them to one another by drinking-vessels.

Applications of sulphate of copper or alum were found to be most efficacious. Boric acid is useless: indeed, the streptococcus develops readily in boric bouillon.[1]

STOMATITIS ACCOMPANIED WITH ULCERATION.

Definition.—Grouped under this heading are the various morbid conditions of the oral mucous membrane which consist essentially of an inflammation proceeding to molecular necrosis of the tissues.

Varieties.—There has ever been a tendency to an over-separation under distinct names of the various forms of ulceration of the mouth, so that it is difficult now, without risk of being misunderstood, to avoid following a similar course. But, as varying aspects of the same morbid process, it should be remembered that these different affections are fundamentally identical, differing, for the most part, in their severity and extent in accordance with the nature of the cause or the constitutional condition of the patient. Although it is not difficult to find what may be called typical cases of the following varieties of ulceration, yet there are many which it is not easy to refer exactly to either group, and which serve to show the essential uniformity of all.

The simplest form of ulcer is that which follows a catarrhal stomatitis, when the death of the superficial tissue-elements has exceeded the coincident repair, with the result of a destruction of surface. What may be regarded as a more intense phase of the inflammatory process, commencing with the formation of vesicles which in a short time burst and leave small ulcers, is represented by the so-called aphthæ. Another variety of the change consists in the formation of what are known as false membranes, which on separation leave an ulcerated surface,—membranous stomatitis; and still another, in a molecular necrosis of the mucous membrane which tends to spread extensively and form pulpy fetid sloughs.

SIMPLE ULCERATION.

The ulceration of the mouth herein included scarcely requires separate mention, except to give completeness to the subject.

Sometimes a simple stomatitis, in place of entirely recovering, ends in a very superficial ulcer of variable shape and extent, on the inner side of the lips or cheeks, or the constant irritation of a broken tooth or the persistent irritation of accumulations of tartar may lead to a similar result in the corresponding part of the cheek or gum.

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[1] Progrès Méd. de Paris, 1886, p. 597.

Such ulcers are of the very slightest depth, are clean and free from grayish or yellowish sloughs, have a reddish and often slightly bleeding surface, are moderately painful, and give rise to scarcely any symptoms beyond those due to the soreness. They rapidly heal when the inflammation has subsided or the cause is removed.

Here, also, may be mentioned those abrasions and excoriations so commonly met with in cachectic children, due to the picking at the lips which they so frequently practise, to an extent sometimes that is almost incredible. For hours, unless prevented, the child will sit picking at the edges and inside of its lips, now and again tearing off a small shred of epithelium, its mouth and fingers covered with blood, and scarcely uttering a sound at its self-inflicted torture. Not infrequently the child will leave the mouth and pick and scratch at some other spot on the body, to return again and tear away the scabs of dried blood which a short respite has permitted the formation of on the mucous membrane. The starting-point of the procedure is frequently a crack or fissure on the lips or at the angle of the mouth, or may be a small vesicle or pustule in that situation. Such wounds are often very obstinate in healing, even if means be taken to prevent further injury, since the reparative powers of the tissues in these children are much deteriorated, and very intractable ulcers may result.

APHTHOUS OR FOLLICULAR STOMATITIS.

Synonyme.—Aphthæ.

Definition.—The words aphthæ and aphthous, derived from ἅπτειν, to "inflame," have been employed with very various significations, even to include the parasitic affection thrush,—with which term, indeed, by some they have been used synonymously,—with the result of much confusion. Their general acceptance renders it undesirable to dispense with them entirely, and it is preferable to limit their application to a form of stomatitis resulting in the formation of small ulcers which run an acute course, tending to recovery, and having tolerably definite characteristics, to be immediately described.

Etiology.—This condition is most common in children between the ages of two and six years,—*i.e.*, subsequent to the first dentition,—and occurs with equal frequency in both sexes.

In very many cases no cause whatever can be ascertained, the ulcers appearing in perfectly healthy children. Dentition would seem sometimes to be responsible, and the direct irritation of objects thrust into the mouth and especially an excess of saccharine food are set down as the determining causes of many others. In another and very numerous class of cases the malady supervenes on a deteriorated state of health, such as is brought about by tuberculosis, measles, scarlet fever, pertussis, or prolonged gastro-enteritis; and it has also been met with in association with herpes of the lips and with impetigo of the face and head.

Occasionally several children in the same house may suffer; and such cases have been spoken of as epidemic, though on no very good grounds.

The climatic changes of spring and autumn appear to be most favorable to the existence of the disease.

Morbid Anatomy.—Those ulcers to which the term aphthous is generally applied are small in size, varying from that of a pin's head to a quarter of an inch in diameter, mostly well defined and clean cut, of fairly uniform round or oval shape, and are almost always discrete and average from six to ten in number. Exceptions to these characters are met with, and a single large ulcer has been described as aphthous, or several small ones may coalesce. Their favorite situation is on the inner surface of the lower lip, close to the frænum, but they are often seen in the furrow between the gum and the cheeks, or on the inner surface of the cheeks and lip or the edges of the tongue, but seldom on the palate or gums.

The ulcers, which are quite superficial and somewhat raised above the level of the mucous membrane, are surrounded by a very sharply defined livid or bright red ring, which contrasts markedly with the general surface of the intervening healthy structure. The floor of the ulcer remains persistently yellowish throughout.

As to the exact mode of origin of these ulcers very different opinions are held, the diversity being, without doubt, due in part to the lack of opportunity of seeing the very commencement, and in part to the difficulty of recognizing the earliest stages of the process on the moist, glistening surface of the mucous membrane. According to some (and with these my own experience is in accord), there first appears a small pearly-gray vesicle very similar to a spot of herpes; whilst others consider that the small area of inflammation becomes in the deeper epithelial strata the seat of an exudation of fibrinous material and leucocytes with fatty granules, the superficial layers shortly becoming torn through and leaving a thick, whitish or yellowish-white, very adherent patch, surrounded by the bright ring above mentioned. The vesicle soon ruptures or the exudation gradually wears off, leaving a shallow ulcer with a grayish-yellow surface, which usually heals in the course of a few days, leaving a temporary reddish spot on the mucous membrane. The mucous follicles are by many (following Van Swieten and later Billard) regarded as the seat of these ulcers, and hence the term follicular stomatitis which is frequently used: the inflammation would then be considered as commencing in the glands, the ducts of which becoming blocked give rise to the vesicular appearance at first noticed.

Fresh spots are likely to appear so long as the cause may exist, while others are in process of healing. "It is exceptional," say Drs. Rilliet and Barthez, "for this malady to be unaccompanied by some gingivitis, which is most marked at the edge of the gums in front, where the mucous membrane becomes red, shiny, swollen, and sometimes bleeding. This sign has often been useful in diagnosing the disease when there has been but a single small ulcer concealed in the gingivo-labial furrow."

Symptoms.—Whether there be any preliminary symptoms—such as fever, heat and dryness of the mouth, thirst, or gastric disturbance—before the appearance of the ulcers it is difficult to say with certainty, but if there be they must be of very brief duration.

The most characteristic symptom of this affection is the extreme pain which the ulcers cause when touched, and hence it is that the child persistently resists being fed, even to such a degree as may lead to positive harm from lack of nourishment unless especial care be taken: those situated on the tongue are especially tender and are the most persistent. There is an increased flow of the buccal secretions, but not to the extent which is met with in some other forms of ulcerous affections of the mouth, unless the aphthæ be very numerous and confluent, and the odor is never offensive.

In the majority of cases the general symptoms are entirely absent or are quite trifling. Slight fever, thirst, a furred tongue, with a sunken appearance about the eyes and variable degrees of irritability, are among the symptoms most generally noticed; but, inasmuch as the malady may manifest itself in the course of a general state of ill health brought about by a previous severe or acute disease, the condition of the child may be more serious, even typhoid symptoms being present. It is less, however, to the aphthæ than to the associated state that such severe symptoms are to be attributed. Gastro-intestinal derangements are especially prone to occur with aphthous stomatitis, but the writer sees no good reason to consider, as is commonly done, that the latter is a result of the affection of the lower portion of the tract, but rather that both are due to some common cause.

Here may be mentioned a peculiar form of ulceration of the mucous membrane which is sometimes met with in the new-born, but affecting only the extremely cachectic and ill-nourished. Described by Parrot[1] under the name of "plaques ptérygoïdiennes," but known also as "aphthes de Bednar," they appear as two symmetrically-placed ulcers on the hard palate close to the velum, one on each side of the middle line. At first shallow and of oval shape and considerable size and with a yellowish base, they may so remain, showing little tendency to heal. But sometimes they extend deeper and involve the bone. They are invariably associated with such a degree of marasmus as to preclude all probability of the infant's recovery.

Another occasional cause of ulceration in the mouth in the very young is to be found in the small collections of epithelial cells known as epidermoid cysts, which have been regarded as glands arrested in development. These densely-packed nodules of cells may break down and form small ulcers, which are generally met with along the margins and raphé of the hard palate. (First described by Guyon and Thierry, 1869.)

Diagnosis.—The appearance of the ulcers and their extreme tenderness are sufficient to enable the disease to be recognized at once. Herpes of the mouth, which might be mistaken for the vesicular stage of this affection, rarely, if ever, occurs without accompanying spots on the lips, and does not produce such well-marked ulcers. Assuming that aphthæ originate in the mucous follicles, their situation alone would distinguish them from herpes or other forms of ulcerative stomatitis.

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[1] Clinique des Nouveaux-nés, L'Arthrepsie, 1877.

Prognosis.—Aphthous stomatitis is not a serious disease, though it often accompanies fatal conditions. Of itself it tends to cure, and unless the cause be very persistent attains that result, but it frequently relapses, and some children appear to be very prone to its recurrence. There is a very common notion that the development of aphthæ (though the word in its connection oftener probably indicates thrush) in the course of any severe illness, or in the state of prostration and enfeeblement which follow thereon, is a sign of approaching death. There do not seem to be any good grounds for such an idea, for even in extreme cases the aphthæ which may have appeared have been recovered from within a few weeks of death, and several attacks may precede the final one. Duckworth[1] considers them to be of gravest import when they extend to the pillars of the fauces and the pharynx; though such conclusions are drawn from cases in adults. In the cachectic and weakly the ulcers may be very slow to heal. The treatment will be considered later.

MEMBRANOUS STOMATITIS.

Definition.—By this term is meant that form of inflammation in which a so-called false membrane is formed in or on the mucous surface, leaving an ulcer when separated off.

Morbid Anatomy.—If the inflammatory process be of special intensity or of a specific character, the products, in place of consisting of a mere increased secretion with proliferated epithelium and leucocytes which is readily thrown off from the surface, may become more coherent and form what is known as false membranes, which exhibit some variety in constitution and considerable difference in their degree of adhesion to the surface. Fundamentally such membranes consist of a fibrinous exudation among the epithelial cells and into the meshes of the mucosa, together with abundant leucocytes and a few blood-corpuscles; this exudation by coagulation involves the tissue-elements, which themselves undergo a form of necrosis described as coagulative or hyaline, the cell-substance becoming coagulated and rigid. In this way plaques of various thickness and extent are formed, sometimes entirely within the surface of the mucous membrane, at other times forming elevated patches of the appearance and consistency of wash-leather. It will be readily seen that on the degree to which the mucosa is involved will depend the greater or less adherence of the false membrane, the corresponding facility with which it may be removed, and the amount of bleeding which such procedure may give rise to. If only the epithelial layers are included, the false membrane will be on, rather than in, the mucous surface, and may be shed, leaving little indication.

The typical false membrane is met with in diphtheria, but the word diphtheritic (meaning a skin) has been applied to similar membranes produced under conditions of inflammation other than those of the specific disease diphtheria, to which the expression diphtheritic should properly be restricted. No practical good is to be gained by attempting to distinguish between diphtheritic and croupous membranes according to the extent to which the tissues have undergone hyaline necrosis; and the word croupous really refers to the character of the breathing when the larynx is obstructed by false membrane, etc., and should not be applied to the membrane itself.

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[1] St. Bartholomew's Hosp. Rep., 1879, p. 18.

Fragments of the membrane may be thrown off *en masse*, being separated by a process of ulceration going on around and beneath it, as would be the case with any other foreign body; or it may undergo disintegration into a granular débris, part of which remains as a coating to the floor of the ulcer. The artificial removal of the membrane is frequently apt to be followed by its re-formation.

Varieties.—Diphtheritic affection of the mouth is treated of in the article on diphtheria (vol. i.). Here it may be sufficient to say that the occurrence of the membranous patches is very rarely limited to the mouth, and very seldom commences there, usually spreading from the tonsils and fauces, and that a micrococcus similar to that met with in the lymphatics, blood, kidneys, and elsewhere is found swarming in the membrane.

Mention has already been made of the view held by some, that aphthæ commence by an exudation into the superficial part of the mucosa and adjacent epithelial layers, forming a small membranous patch which gives place to an ulcer.

In contrast to such forms of membranous stomatitis are those cases where the superficial layers of the oral mucous membrane perish from the application of caustics or boiling fluids. Areas of the surface to varying depth and extent are completely and suddenly killed, forming opaque or yellowish-white patches of coagulated tissue which on separation leave an ulcer. Here the necrosis starts from the surface and extends inward, not, as in the former cases, commencing in the mucosa and thence spreading outward.

ULCERATIVE STOMATITIS.

Synonymes.—Stomacace, Putrid sore mouth.

Definition.—A form of inflammation of the buccal mucous membrane which speedily results in extensive ulceration especially of the gums, accompanied with much fetor of the breath, frequently contagious, and with some grounds for believing that it is dependent upon a specific germ. It is rarely fatal, and readily yields to treatment.

Etiology.—The disease occurs with about equal frequency in each sex.

It is far more common after the second year,—that is, after the completion of the first dentition,—and up to the age of six or seven years, when the second dentition is established. Of two hundred and six cases collected by Rilliet and Barthez between the ages of two and fourteen years inclusive, one hundred and thirty-three occurred from the third to the seventh year, the number subsequently declining year by year.

The development of the malady is very considerably predisposed to by unfavorable hygienic surroundings; bad feeding, both insufficient in quantity and improper in quality, want of proper ventilation in the dwellings, lack of clothing, neglect, and dirt are all responsible for bringing the children to that depraved state of health in which ulcerative stomatitis is most likely to supervene. But in the absence of such conditions and among the well-cared-for the affection is of frequent occurrence when the bodily state has been deteriorated by severe or prolonged disease, and there is scarcely any malady of childhood that it may not follow on. Duckworth[1] has pointed out the association of the affection with congenital heart-disease, and refers to cases which are identical, so far as the mouth-state is concerned, with the disease now under consideration.

Although met with at all seasons, it is more common in the damp weather which characterizes spring and autumn.

Many cases (eighty per cent., according to Rilliet and Barthez) are attributable to local conditions,—that is to say, which favor decomposition in the oral cavity and are due to want of cleanliness. Such especially are caries of the teeth, accumulations of tartar round their necks, and of particles of food between them; and any circumstances, such as paralysis or toothache, which, by hindering the movements of the mouth, allow fragments of the ingesta to lodge there. It is doubtful, however, whether either of these circumstances is the real cause of the disease, and there is good reason to believe that this is to be found in some form of microbe, for it is well recognized that the affection is very commonly contagious (Bergeron proved it to be so on himself) and is apt to occur in epidemics, while in some places of unsatisfactory sanitary character it may be said to be endemic. Such circumstances as these suggest the probability of an organism being the *vera causa*, the conditions above mentioned being such as to render the oral mucous membrane a suitable nidus for the development of the same. Some, indeed, regard the malady as corresponding to the foot-and-mouth disease of cattle, milk being the vehicle of contagion: of this view, however, there is no proof at present. Dr. Payne[2] is inclined to believe that the virus is the same as that which causes impetigo contagiosa, for he has, in common with other observers, frequently noticed a pustular eruption on the lips and hands accompanying ulcerative stomatitis, and he considers that an ulcer in the mouth corresponds to a pustule or excoriated patch on the skin, the formation of crusts in the mouth being obviously impossible, and all inflammations in the mouth tending to ulceration.

The ulceration of the mouth which often follows mercurialization (though far less common in children than in adults) is a typical form of ulcerative stomatitis. Lead, copper, and phosphorus may lead to the same condition.

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[1] St. Bartholomew's Hosp. Rep., 1879.

[2] St. Thomas's Hosp. Rep., 1883.

Lymphoid overgrowths in the submucosa of the gums may break down and lead to ulceration of the mucous membrane.

Morbid Anatomy.—That form of ulceration of the buccal mucous membrane which is specially designated "ulcerative stomatitis" is rarely observed before the actual destruction of tissue has taken place, and any description of a pre-ulcerous stage is therefore conjectural. There are presumably a hyperæmia and an exudation of inflammatory products into the mucosa, but in the condition now described there is no agglutination of these products with the tissue-elements (epithelial and fibrous) into distinct false membranes. In part perhaps from the pressure exercised by the effusion, and partly perhaps from the specific nature of the inflammation, a molecular necrosis takes place, which rapidly results in the formation of an ulcer.

In by far the greater number of cases the process commences at the alveolar margin of the gums opposite the lower incisor teeth, and from there extends backward towards the molars, passing over to the inner side of the jaw in any interval that may exist in the dental arch. Sometimes (in forty-eight out of three hundred and thirty-nine cases quoted by Rilliet and Barthez) the ulceration is limited to the gums, but generally while restricted to one side of the mouth it extends to the contiguous surfaces of the lips, cheeks, and edges of the tongue, whilst the hard and the soft palate and the dorsum of the tongue escape.

The gums which are the seat of the affection are puffy and swollen, but neither tense nor hard, are red or of a livid purple, and the ulcerated surface which reaches up to the necks of the teeth is covered with a grayish or yellowish-gray layer of pulpy granular sloughs, from which bleeding takes place, some purulent fluid exuding from the alveolar sockets on pressure. On the inner surface of the lips and cheeks, usually at points corresponding to the affected gums, the ulcers may attain considerable size, with irregular outline, but with no special thickness of the edges, and are covered with a pultaceous débris of necrosed tissue. Frequently, instead of one single ulcerated patch, there are several smaller ulcers separated by healthy tissue. The general swelling of the mucous membrane gives a fallacious appearance of depth to the ulcer, which rarely, however, extends so deeply in this situation as it does on the gums, where it may even invade the bone, producing necrosis, and very frequently so loosens the teeth as to lead to their falling out.

Microscopic examination of a section of the ulcer exhibits a necrotic condition of the tissues extending to variable depths below the sloughing surface, with abundant leucocyte-infiltration and indistinctness of the normal tissue-elements. Throughout this same region swarms of micrococci and bacteria are to be found, but not the characteristic bacilli discovered by Mr. Lingard in noma.[1]

The tongue is swollen and coated, and indented with the teeth, impressions of which may mark the puffy lining of the cheeks.

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[1] See case described by J. Hutchinson, Jr., Path. Soc. Trans., 1887, vol. xxxviii. p. 127.

Occasionally aphthæ are seen associated with this condition.

The submaxillary glands on the affected side are often moderately swollen, hard, and tender, but they do not proceed to suppuration; and the subcutaneous tissues of the cheeks and beneath the jaw are frequently œdematous.

As recovery takes place the surface of the ulcers becomes cleaner, and the bleeding diminishes as the epithelium is gradually re-formed. An irregularity of surface or a very imperfect scar may mark the situation of the ulcer for some time.

Symptoms.—When this affection develops in the state of ill health produced by bad hygienic surroundings or a severe illness, the general symptoms are probably involved in those of the existing cachexia, and even in those cases which may be regarded as primary, or at least due to some local cause, there may be very little beyond local symptoms. The degree of fever is very variable, sometimes reaching 102° or 103° F., but frequently scarcely above normal, and the temperature when raised is marked by no regularity of course. That the pyrexia sometimes terminates by lysis with an accompanying improvement in the state of the tongue and mouth is regarded by Dr. Goodhart[1] as suggesting "that possibly some cases, at any rate, might be due to a specific germ."

Locally there is an excessive flow of acid buccal secretion of extremely fetid odor, sometimes even gangrenous, whence the older name of "putrid sore mouth;" and this will be the case even when the extent of ulceration is very moderate. Pain and tenderness in the mouth exist to such a degree as to interfere with mastication and very often with the proper taking of nourishment. Occasionally bleeding from the tumid ulcerated gums may be considerable, and there is usually a slight hemorrhage which dribbles from the mouth with the saliva on to the pillow.

A diarrhœa with very offensive evacuations complicates some cases, and may be very troublesome, or even serious; it is probably due to the swallowing of the decomposing discharges from the mouth; and nausea or vomiting, when present, may arise from the same cause.

Diagnosis.—Examination of the mouth at once reveals the condition, which cannot well be mistaken for any other. The fetor of the secretion from the mouth is alone sufficient to distinguish the disease from the aphthous and other forms of ulceration, whilst the character of the ulcerated surface and its usual rapid yielding to treatment differentiate the malady from the blackened, sloughing, gangrenous appearance presented by noma, the other oral affection accompanied by fetor.

Prognosis.—Ulcerative stomatitis is not of itself a fatal disease, although death may ensue from the general state of malnutrition which has favored its occurrence. It tends to spread rather than to heal, and may therefore last a considerable time unless it be subjected to treatment, to which it is almost always readily amenable. It is liable to recur if its predisposing cause be not completely removed or the treatment be not sufficiently long continued. Its relation to cancrum oris, into which it is said sometimes to pass, will be referred to under the description of that disease.

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[1] Diseases of Children, 1886.

Treatment of Ulcerous Stomatitis.—The treatment of the various forms of ulceration of the buccal mucous membrane may very conveniently be considered at one time, since the general aim—namely, to remove the cause and to favor the healing process—is the same in all cases, and for the most part is accomplished by the same means.

The general tendency of all these ulcers, except in the most extreme cachectic states, is towards healing, and, provided the source of irritation be removed, this will very frequently be accomplished in a few days without any further treatment than attention to general hygiene and diet. In the majority of cases, however, some therapeutic measures are called for beyond the careful observance of these conditions.

Locally it is of primary importance to maintain all possible cleanliness, whether as a preventive measure by keeping the teeth well brushed and free from tartar and by removal of all remains of food, or by frequent washing of the mouth, especially after nourishment has been taken. The most effective washes for this purpose are a chlorinated water, and solutions of permanganate of potassium (four grains to the ounce), carbolic acid, or boric acid (five grains to the ounce). When the gums are spongy and bleeding, astringents are indicated, such as alum, tannin (glycerole of tannic acid), or rhatany, of various strengths: these are best applied to the surface with a brush.

For promoting the actual healing of the ulcers caustics are of doubtful value, and the solid nitrate of silver or sulphate of copper should rarely if ever be applied,—certainly not when the child is very ill nourished, as the destruction they produce only increases the mischief. The small irritable aphthous ulcer close to the frænum of the lip may sometimes be touched by the caustic sufficiently to produce a very slight superficial slough, under which healing takes place, but this treatment is inapplicable when the aphthæ are numerous and inadvisable when they are extensive; in such cases solutions of these salts (gr. x ad ℥i) may be usefully employed.

Fortunately, we possess in the chlorate of potassium or of sodium a drug which has been rightly spoken of as a specific in almost all forms of ulceration of the mouth. Its efficacy is undoubted, and improvement speedily follows its use. It was first employed for these maladies by Dr. Hunt,[1] but its general use was specially due to the advocacy of Dr. West. It is most effective when given internally in amounts of twenty to sixty grains daily, in divided doses, with a few drops of glycerin or syrup according to age. Owing to its rapid elimination by the buccal glands, it is kept constantly in contact with the ulcerated surface and more continuously and effectively than if it be only used as a wash, although, if the child be able completely to rinse the mouth out with a solution of ten grains to the ounce, this may be sufficient; or there is no harm in combining both the internal and external administration. The application of the powdered salt to the ulcerated surface may be adopted, especially when swallowing is difficult. Although it would seem to be a common error to give too small doses, it should be remembered that the drug acts in poisonous amounts by converting the hæmoglobin into methæmoglobin, and in quantities short of such a result does, if its use be prolonged, tend to produce anæmia. The application of dry calcic chloride has been known to succeed in the rare cases where the chlorates have failed. Borax is not nearly so useful in these affections as in thrush, though it is often given in combination with the chlorates.

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[1] Med.-Chir. Trans., 1823, vol. xxvi.

The extreme painfulness of some of these ulcers, particularly the aphthous variety, which is intensified by the contact of food, requires treatment, and this may be effected by painting the mucous membrane with weak (five per cent.) solution of cocaine before food is given, or by using in the same way such demulcent and soothing applications as the mucilage of sumach (prepared from the inner bark of Rhus glabrum), which has been highly extolled for this purpose by Dr. Corson, or decoction of marshmallow or mucilage of quince. The action of these is essentially protective to the raw, painful surface, as well as somewhat astringent.

The need for proper feeding is paramount, and what I have already said as to the value of alcohol when describing the treatment of simple stomatitis equally applies here.

Tonics are very frequently necessary, and some of these may conveniently be combined with the chlorate of potassium. Such are quinine, gr. ss to iss; solution of perchloride or of pernitrate of iron, ♏iii to ♏v of either, potassii chloratis, gr. iii, glycerini, ♏xv, aquæ ad ʒii; and cinchona and ammonia: this latter may be given in the form of—

℞ Spiritus ammoniæ aromatici, ♏v to ♏xv;
Ext. cinchonæ liq., ♏iii;
Infus. cinchonæ ad ʒi vel ʒii, according to age:

or from three to six minims of dilute hydrochloric acid may be substituted for the ammonia.

CANCRUM ORIS.

Synonymes.—Gangrene of the mouth, Noma.

Definition.—A malady of infrequent occurrence, and usually secondary, consisting of a rapidly-progressive necrosis of the cheek or gum, which is commonly fatal, and is recovered from only with permanent loss of tissue.

Etiology.—The disease is by no means common, and is even less so now than formerly. At the East London Hospital for Children, which is situated in a very poor and densely-crowded district, during the seven years 1881–1887 inclusive only five cases occurred, with a total number of six thousand three hundred and sixty-four admissions during that time; and at the Hospital for Sick Children, Great Ormond Street, during the thirteen years 1876–1888 only six cases occurred, with a total admission of upward of thirteen thousand patients.

Certain conditions appear to exercise very considerable predisposition towards the development of this terrible malady.

All records go to show its greater frequency in females: of the one hundred and three cases collected by Rilliet and Barthez sixty-three were girls, a circumstance which finds very insufficient explanation in the alleged greater weakness of this sex in childhood.

It is distinctly more prevalent between the ages of two and five years, more than half the cases occurring during that time, and, although rare cases are met with in adults, it is especially an affection of early childhood.[1]

Such mal-hygienic surroundings as tend to the deterioration of the children's health would seem to favor its occurrence, for it is certainly more often seen when there is overcrowding, with all that that implies; but it is by no means confined to children in such circumstances.

It has been regarded as almost endemic in low-lying, damp countries, such as Holland and parts of Sweden; but it is not contagious. Fewer cases are recorded in summer and in winter,—spring and autumn, when the exanthemata are rife, being the favorite seasons.

The effect of previous disease, both by producing general impairment of health and probably also by some specific influence, is undoubted in bringing about noma. Of all antecedent maladies measles is by far the most potent, it having been noted as a precursor in more than half the recorded cases. Far less frequently has cancrum oris supervened on scarlet fever, pertussis, varioloid, and typhoid. Among the poor, half-starved native children in India it is a common sequence of malarial fevers (Goodeve). The influence of measles is partly but not entirely explained by the great frequency of stomatitis in that affection.[2] How far stomatitis with ulceration independent of measles is a determinant of gangrene of the mouth is very doubtful, although rare cases are so reported; and the same may be said for mercurialization as a cause.

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[1] An interesting case of cancrum oris associated with erysipelas in a man aged forty-seven, which proved fatal, is recorded by Dr. Brydon, of the Hartford Hospital, Paris, in the British Medical Journal, 1882, vol. ii. p. 838.

[2] It is not easy to see any connection between the greater prevalence of the disease in girls and its marked association with measles. Although scarlet fever is said to be commoner among females at all ages (Dr. Whitelegge, Trans. Epidem. Soc., 1888), I am not aware that the same can be said of measles, and, on the other hand, the Registrar-General's Reports show a preponderance of deaths from measles among males at all ages over females.

The tendency to gangrene engendered by the acute specifics does not always show itself by cancrum oris: the septic state which characterizes these diseases, instead of being recovered from, would appear to produce so profound a change in the nutrition of the tissues as to cause their death *en masse;* but what determines the preference for the cheeks or the vulva, or more rarely for the limbs, is quite unknown.

Morbid Anatomy.—The structures involved in this serious disease are those of the cheeks and the adjacent gums; occasionally the latter alone are affected, and still more rarely is it confined to the buccal walls or the lips. It attacks either side with equal frequency, and sometimes (eleven of one hundred and three cases) both sides of the face are implicated. The fold between the lips or cheek and gum is a frequent starting-point, whence it spreads both towards the surface and to the jaws; many cases commence close to the angle of the mouth.

The comparative rarity of the disease, together with the extremely rapid progress of the morbid changes which usually lead to the lesion being well established before it comes under observation, have permitted difference of opinion as to the exact site of commencement and the actual nature of the initial departure from the normal. Some observers especially insist that the mucous membrane is first affected, while others describe an induration in the thickness of the cheek as the first change, the mucous membrane of the skin being subsequently involved. Although Henoch[1] refers to a case of cancrum oris which developed from a phlegmon in the cheek and did not invade the mucous membrane, it may be taken as an almost universal rule that the mucous membrane is affected and that this disease does not commence in the skin, thus offering a point of distinction, among others, between noma and malignant pustule.

The usual course of events is as follows. On the surface of the mucous membrane, preferably in one of the situations above indicated, is first seen a dark-gray, ragged, sloughing surface, covered with a sanious, offensive discharge; preliminary to this an ichorous bleb has been described, but it is excessively rare for the case to be seen at this stage. The duration of this ulcerous condition may be not more than two or three days, and its earliest appearance is not always such as to indicate with certainty the impending grave change. Very soon, however, a definite, circumscribed, hard, and slightly tender nodule is perceptible in the substance of the cheek, corresponding to the position of the ulcer, and in a short time—within twenty-four hours, as a rule—the skin over this induration becomes of a bright red, soon turning to a livid purple, tense, hot, and greasy-looking. Progressive with this extension of the disease in the thickness of the tissues is an extent in its area: the destruction of the mucous membrane spreads over the surface and to the adjacent gums, the brawny swelling invades more and more of the substance of the cheek, and the discoloration of the skin extends wider and wider. The whole cheek and side of the face become more or less tumid, and this may reach to the eyelids and downward to the neck. The cutis over the dark livid area, which is gradually deepening in color until it becomes almost or quite black, peels off, previously having been raised into a bulla, soon to be followed by the characteristic appearances of gangrene; a black dry or very slightly moist eschar is rapidly formed, and spreads over the already indurated, swollen cheek, extending meanwhile in depth to meet the similar destruction which has been proceeding outward from the mucous surface. The eschar may be formed as early as the second day, or perhaps not until the end of the second week, but it usually appears on the third to the seventh day. Sooner or later, chiefly dependent upon how long the child may live, these foci of necrosis meet, and a mass of dead tissue extends from the mouth to the surface. The moist character of the mucous membrane favors a constant removal of the débris in flakes and shreds, which are frequently swallowed, while the separation of the cutaneous slough may be postponed, though finally coming away and leaving a jagged, unhealthy, fetid wound, which joined with the similar one in the mouth establishes a perforation of the cheek. The extension to the gum quickly involves the bone, sequestra of which separate, and the teeth loosen or drop out. The process is one of rapidly-spreading gangrene, commencing probably in the mucous membrane, extending thence in every direction, and involving every tissue, showing little tendency to limitation, but, if life last, invading chin, eyelids, nose, and even ear, internally exposing the bones of the upper and lower jaw and sometimes the nasal fossæ, but very seldom crossing the middle line or extending below the lower border of the jaw. In the less severe cases the eschars on the mucous and cutaneous surfaces may not join, but remain separated by the adipose and muscular tissues of the cheek, infiltrated with serum, but not gangrenous.

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[1] Diseases of Children, 1883.

In the course of the disease the vessels, for the most part, escape destruction, though the arteries become plugged with firm clots throughout the length of the sphacelus, and thus hemorrhage is almost entirely prevented. The nerves also exhibit considerable resistance to the mortification, retaining their structure almost intact. The duct of Steno has been known to remain permeable in the midst of a mass of completely necrosed tissue.

Notwithstanding the serious change in the parts involved, the submaxillary lymphatic glands frequently remain normal, and are never more than slightly enlarged and soft, suppuration in them being unknown.

The occurrence of aphthæ as a preliminary to noma has been noted, but there are no grounds for assuming more than accidental coincidence.

The condition of the blood has long been of considerable interest in cancrum oris, especially in its bearing on the pathogeny of the disease. Dr. Sansom in 1878[1] first described with any fulness certain moving bodies found in the blood of a girl aged four and a quarter years, the subject of a typical noma which proved fatal in eight days. During life the white corpuscles were seen to be excessive in number, very granular, and unusually active; the red showed marked tendency to form rouleaux, and varied considerably in size,—from $\frac{1}{6000}$ to $\frac{1}{3200}$ inch in diameter,—as is the case in idiopathic anæmia. In addition there were numerous granules, and also numerous "small, highly-refractile bodies, endowed with powers of rapid locomotion; each one refracted the light in such a manner that a small bright cross was visible in its substance. Thus they resembled crystals of oxalate of lime, with the exception that they were not perfect octahedra." The movements were not of the so-called Brownian character, but distinctly resembled those of bacteria, and were stopped by such reagents as quinine and carbolic acid, whilst heat as well as dilute solutions of potash or of sulphuric acid increased the activity of the particles. It was estimated that twenty occupied the space of one full-sized red corpuscle. When the temperature of the patient was high these bodies were most numerous and were aggregated into zooglœa masses. On the day of the child's death ordinary bacteria were seen in the blood in addition. The urine and fæces examined immediately after being voided contained similar translucent motile particles, as also the discharges from the wound on the face, together with vibriones and bacteria of putrefaction. Inoculation of animals with blood taken from the heart post mortem produced fatal septicæmia, and motile bodies similar to those above described were found in the blood of the animal; but this was not the case with inoculation of matter from the gangrenous cheek, which appeared to produce no effect. (See Plate, Fig. 1.)

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[1] Trans. Royal Medico-Chirurg. Soc. Lond., vol. lxi.

Dr. Morse,[1] referring to a former expression of opinion that moving bodies might be seen in the blood in this disease, describes a fatal case in a boy aged eleven years, where the lesion was of considerable extent, involving cheek and both jaws as well as the adjacent tongue. Frequent examination of the blood demonstrated refractile, pseudo-crystalline, motile bodies such as described by Dr. Sansom, with which they corresponded in behavior with reagents and in conditions of appearance.

The occurrence of these strange objects is not invariable in noma: Dr. Sansom has met with cases in which they were entirely absent, and he suggests that they are the result of cultivation in the blood of less virulent forms of organisms derived from the sloughing wound, a view that would regard them as a result and not a cause of the gangrene. They were not observed in a case of gangrene of the vulva in an adult.[2] As Rilliet and Barthez point out, it is necessary, in assigning to any organisms found in the blood in this disease their proper share in the production of the malady, to remember that it is so constantly secondary to other maladies which are attributable to specific living forms that the previous condition of the blood has to be reckoned with.

A valuable contribution to the pathology of this affection has more recently been made by Mr. Alfred Lingard.[3] He found in cases of noma

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[1] New York Medical Record, January, 1885, p. 37.

[2] Dr. Herman, Trans. Obstet. Society, 1883.

[3] Lancet, 1888, ii. 159.

DESCRIPTION OF FIGURES.

Fig. 1.

Blood from a Case of Cancrum Oris (after Dr. Sansom).—*a*, white corpuscles; *b*, red corpuscles, showing great variations in size; *c*, translucent motile bodies; *d*, aggregations of the same: × 450 diameters.

Fig. 2.

Section through Line of Advance of Noma, from cheek of a child.—*a*, healthy tissue,—muscular fibres, fat-cells, and artery in section; *b*, the tissues shrivelled and necrotic from the action of the bacilli; *c*, line of bacilli advancing: × 120 diameters.

Fig. 3.

Bacilli Noma, from the line *c–c* of Fig. 2: × 450 diameters. [Exactly similar bacilli, producing similar changes in the tissues, are found in a form of ulcerative stomatitis in the calf and in the pig, and in a form of pneumonia in the hare.]

Figs. 2 and 3 were drawn from specimens prepared by Mr. A. Lingard, M.B., and kindly lent to me by him to illustrate the quotations from his paper on page 979. They have never before been published.

Fig. 4.

Saccharomyces, or Oidium Albicans, artificial cultivation (after Grawitz).

Fig. 5.

Scraping from a Patch of Thrush, showing scaly epithelium, spores, segments of the fungus, and granular débris.

Fig. 1

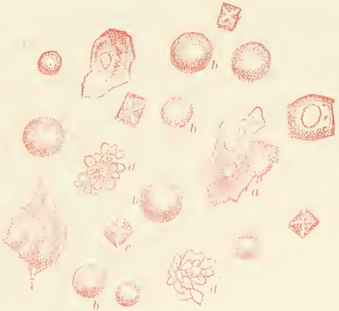


Fig. 2

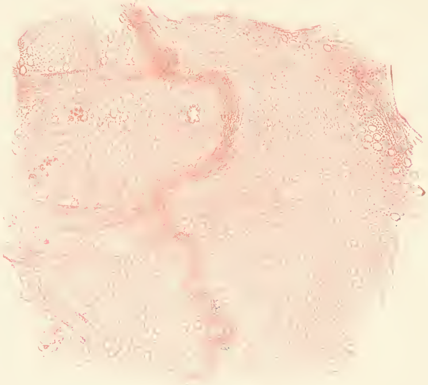


Fig. 3.

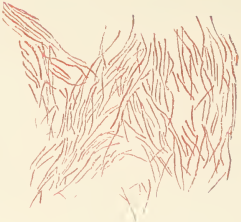


Fig. 4.

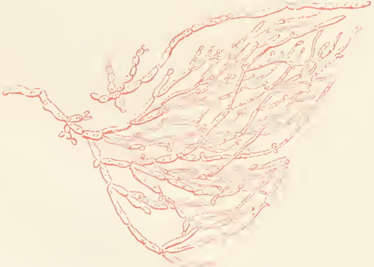
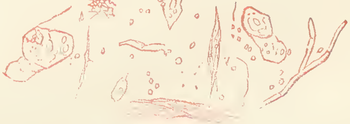


Fig. 5.



in the human subject, and also in a form of ulcerative stomatitis affecting calves, a peculiar species of micro-organism consisting of "long thread-like growths, the individual threads being made up of small bacilli varying in length from 0.004 mm. or less to 0.008 mm. or more, and about 0.001 mm. in thickness. These organisms were found in great numbers at the line of extension of the necrotic patch." Cultivations of these organisms and their subsequent inoculation into other animals gave rise to "appearances precisely similar to those seen in the original disease." Among the most remarkable appearances are those seen in the walls of the heart, a section of which discloses numerous circular necrotic foci which consist of clusters of these thread-like organisms lying in the tissues, all structural characteristics of which had disappeared. Surrounding the patch was an inflammatory zone containing abundant leucocytes. "In many places were found these organisms in varying numbers, infiltrating the intermuscular tissue and surrounding the capillaries and lymphatics. In these cases they appeared like long bundles or leashes, of circular contour, passing along the lumen of the vessel. In the human heart, in consequence of the early death of the patient, such serious lesions are not met with; in the five necropsies made on children dying of noma only once was found heart-lesion, and that was characterized by the presence of petechial spots, from ten to twelve in number, of a dark-red color, dotted over the surface of the heart immediately beneath the exocardium. On closer examination these spots were found to be slightly elevated above the surrounding tissue. Microscopically these petechial elevations presented a small hemorrhage raising up the exocardium, whilst the apposed portion of the muscular bundles of the heart was surrounded by a varying number of blood-corpuscles. On very careful examination I was able to verify the presence of organisms—always micrococci or diplococci—which had gained entrance to the circulatory system from the grumous material always found in the cavity of the mouth, etc., in such cases. In no instance was I able to discover the thread-like growth or bacillus nomæ." (See Plate, Figs. 2 and 3.)

Post-mortem examination invariably discloses grave alterations in various viscera, some of which appear to be directly due to septic matter conveyed from the mouth. Thus, both lungs are generally (ninety-five per cent. of the cases) the seat of a diffused broncho-pneumonia, especially towards the bases. The infected particles may be assumed to be carried along the air-passages from the oral cavity or in the branches of the facial and other veins to the lungs *via* the heart.

The next most frequent lesion is a form of colitis characterized by patches of grayish membranous material with intervals of hyperæmic mucous membrane. Dr. Wharton[1] describes an interesting case of this condition showing the patches to be due to infiltration of the mucosa with round cells and granular matter, the normal constituent tissues being no longer recognizable.

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[1] Med. and Surg. Reporter, Philadelphia, 1887, p. 73.

The cardiac changes as described by Mr. Lingard have already been referred to.

The kidneys may exhibit the appearances of acute nephritis; and Wilks described the liver as fatty in two cases.

The post-mortem appearances of pleurisy, purulent pericarditis, or peritonitis may also be met with. Gangrene of the lungs, skin, genitals, or extremities may coexist with cancrum oris.

A peculiar form of gangrene limited to the gums has been described by Klementowsky, of Moscow,[1] as seen by him in three cases only during twenty years' experience, and named osteo-gingivitis gangrænosa neonatorum. The ages of the infants were six, thirty-eight, and forty-five days respectively, and two were males. The condition was very similar in each, and all died in the course of a few days. The attack commenced with high fever, soon followed by a purple swelling of the gums on one side, which ended in gangrenous abscess and ulceration, the teeth falling from their sacs and the bones becoming necrosed; purulent peritonitis developed in one case.

Symptoms and Course.—Associated with the destructive process which constitutes the most characteristic feature of the disease is an extremely pungent fetid odor from the mouth, even before any actual gangrene is obvious, and it may, indeed, be the very first circumstance to call attention to the condition. As the necrosis extends the fetor becomes almost unbearable, though apparently far less offensive to the patient than to those around. The flow of saliva is increased, and soon becomes sanious and thick from the decomposing discharges from the wound. Previous to the appearance on the skin of the livid patch, the affected side begins to swell, and, except at the site of the gangrene, is usually pallid. In some cases the little patient is prostrated from the first, and appears as if knocked over by the disease; but far more commonly the child appears unconcerned and continues playing with its toys, at least for a few days, notwithstanding the advance of the destruction, and seldom complaining of pain. Sometimes the child is restless and irritable, sleeping but little, and finally becoming delirious. The degree of fever is not high; the temperature may reach to 104° F., but more often does not exceed 103°, and falls to normal several times during the course of the malady. The pulse is frequent, small, and quick, and the respiration hurried, with exaggerated movements of the alæ nasi. The skin may be dry or moist, and the extremities are apt to be cold. There is frequently œdema of the feet, and sometimes the œdema may be more extensive. The appetite may remain good almost to the last; the tongue, which is blackened towards the affected side, is elsewhere moist and somewhat coated.

Diarrhœa is of common occurrence, whether from the irritation of the putrid matter which is swallowed or from a coexistent colitis; vomiting, however, seldom happens.

It is commonly the case that there is a broncho-pneumonia, though the symptoms may not be marked, and may be almost overlooked in the presence of the terrible condition of the mouth. How far this complication is a septic manifestation, or whether the original measles may be responsible for it, is not always clear. Owing to the plugging of the vessels in the necrosed area, hemorrhage rarely occurs.

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[1] Quoted in Lond. Med. Record, 1879, p. 496.

The fatal cases seldom last much beyond sixteen or eighteen days, and may not exceed half that time. When the prostration is very profound the child may die before perforation of the cheek has taken place, or, indeed, before the cutaneous eschar has been fully formed. Convulsions may precede the end, but, as a rule, death supervenes quietly.

When recovery takes place, whether before or after destruction of the cheek, the wound when the slough separates gradually assumes a healthier appearance, instead of the ashen-gray, indolent, mortifying aspect which characterizes it in the progressively fatal cases. Granulations spring up around the edges and on the surface, and healing finally takes place after some months by the formation of fibrous cicatricial tissue, which, only partly filling the cavity, leaves much deformity, sometimes with the establishment of such adhesions between cheek and gums as considerably interfere with the proper opening of the mouth. The disease has been known to relapse even more than once; Rilliet and Barthez record a case in which it did so five times, the boy, aged eleven years, finally recovering. On the other hand, cases which are seemingly proceeding to cure may suddenly assume a grave aspect and prove fatal.

Diagnosis.—The disease proclaims itself, and cannot when fully established be mistaken for aught else. Reference has been made to its similarity in appearance to anthrax, but, apart from the fact that the latter usually occurs in adults exposed to a known contagion and does not commence in the oral mucous membrane, the specific bacillus anthracis may be detected in the fluid discharges of the wound and in the blood. The relation of ulcerative stomatitis to cancrum oris is one of interest not only from a causal but also from a diagnostic point of view. That very rarely cases of ulcerative stomatitis do pass into a condition of gangrene has been already mentioned, but the foregoing account of the morbid changes in the latter disease will serve to show the essentially different character of the two processes. Extreme neglected cases of ulceration may bring about considerable destruction of surface and may even lead to some necrosis of the bone, but the loss of substance is never considerable and does not involve in gangrene the integuments, which remain normal; and, again, it is rarely fatal. The characteristic appearances of the blood, moreover, are wanting in stomacace, though, as Mr. Lingard has shown, identical organisms are found in the ulcerative stomatitis of calves and in noma in children. On the view that cancrum oris is but one local expression of a general morbid state, it may be that the stomatitis, whether simple or ulcerous, is an important factor in determining the locality of the gangrene.

Prognosis.—Although an extremely fatal malady, noma is not invariably so. Statistics differ much as to the actual percentage of recovery, but the mortality is approximately seventy-five per cent. Those cases do best in which the necrosis is confined to the gums, but, on the other hand, perforation of the cheek is not necessarily fatal. Absence of chest-symptoms and absence of diarrhœa are hopeful signs, since when they exist to any degree death may be as much due to them as to the mouth-state. When delirium sets in, or when the case is marked throughout by severe prostration, the chances of recovery are extremely slender. Speaking generally, the extent of the destruction is the most reliable ground on which to base a prognosis.

Treatment.—When once the character of the disease is recognized, the indications for its treatment are: first, to arrest the spread of the destructive process; secondly, to prevent general infection, and especially the development of broncho-pneumonia and diarrhœa; thirdly, to maintain the general strength of the patient; and, lastly, to promote the healing of the wound.

To meet the first requirement, we must aim at destroying the virulent morbid action which is present, and substitute for it a healthy process of recovery. For this purpose caustics of all kinds and degrees of potency have been employed: the actual cautery (Paquelin's or the galvanic), fuming nitric acid, solution of bromine, the strong solution of perchloride of iron, solid chloride of zinc, nitrate of silver, chloride of calcium, butter of antimony, Vienna paste, and saturated solutions of iodine in tincture of iodine are but a few of the many escharotics which have been used, and frequently with success. Some practitioners are in favor of an early resort to these strong measures, supporting their view on the ground that the further the disease is allowed to spread the greater the danger of septic infection, and that noma is a disease in which (like anthrax and, some would say, syphilis also) the local lesion exists for a relatively considerable time before general sepsis appears, in which period the best chance for destroying the disease exists. It is certain, however, that if this plan of treatment be adopted the application should be thorough and complete at first; short of that, the irritation induced may only favor the spread of the disease. It is very desirable, also, that the range of application should be entirely under control, and the caustics should not be permitted to destroy beyond the necessary limits: this is very difficult to avoid when fluids are used, however excellent they may otherwise be, and even the various forms of paste recommended are open to a similar objection. For this reason, the actual cautery is by far the best: the operator can see exactly what he is doing, and its power of destruction is complete and immediate. To insure the most effectual application, it is desirable to cut away all the dead slough before using the caustic, or, if the case be in the early stage before the eschar is formed, to incise the swelling and so permit the agent to effect its purpose completely. It should be remembered that the mucous surface must be treated in the same way as the external wound; and here again the cautery is far more convenient. An anæsthetic should be administered. Repetition of the cauterization may be necessary.

Other physicians advise milder applications, at least to commence with, before having recourse to the more heroic treatment, and point to successes with them in justification of their plan. A mixture of sulphate of copper two drachms, and powdered cinchona bark one-half ounce, in four ounces of water, to be applied over the wounds twice a day, is highly recommended by Dr. J. Lewis Smith, of New York. Even such mild remedies as chlorate of potassium or subnitrate of bismuth, powdered over the surface two or three times daily, have been advocated, combined with painting the gangrenous surface in the mouth with a two-per-cent. solution of resorcin. Whichever of the above means be adopted, it is needful to supplement them with antiseptic applications, if only to overcome the pungent fetor: dusting the surface with powdered charcoal, iodoform, or salicylic acid, or lotions of carbolic acid ten per cent., of chlorinated soda, of eucalyptol, of terebene, or of Condy's fluid dabbed over the surface, or more conveniently administered as a spray, are suitable for this purpose.

For its local action chlorate of potassium is given in ten-grain doses every five hours, and Dr. West refers to cases cured by this means together with good feeding and without any use of caustics.

Inasmuch as the pulmonary and intestinal symptoms are largely due to putrid matter which reaches the lungs and the stomach from the mouth, the child should be so placed in bed as to permit the freest evacuation of the discharges and to avoid as far as possible their entering into trachea or gullet; it has even been suggested that tracheotomy should be performed, so that the air which enters the lungs shall not be contaminated by passing through the mouth.

Every effort must be made to sustain the child's strength by good food, wine, and tonics. Strong beef tea, eggs, milk, minced and pounded meat, and farinaceous food, should be given day and night at frequent intervals. Often no difficulty is experienced in giving nourishment, but sometimes, from the prostrate condition of the patient, it may be almost impossible, and feeding by the nose or by nutrient enemata and suppositories should be resorted to. Mixtures containing quinine and iron should be given regularly every four to six hours.

To clean the wound and remove the sloughs, whether caustics be employed or not, charcoal poultices or boracic-acid fomentations are convenient, and when the surfaces are showing indications of granulation this may be encouraged by lotions of boracic acid (twenty grains to the ounce), chlorate of potassium (ten grains to the ounce), sulphate of zinc (two grains to the ounce), or tincture of myrrh, or by an ointment composed of two drachms of balsam of Peru and one ounce of vaseline. Care should be taken during the slow process of healing that the tissue which closes the wound does not form such adhesions as may interfere with the proper opening of the mouth; but, owing to subsequent contraction of the cicatrices, it is impossible to avoid considerable deformity, which is only partially remediable by plastic operations.

SUPPURATIVE INFLAMMATION OF THE GUMS.

Synonymes.—Gumboil, Parulis.

This common affection, which is more fully treated of elsewhere under the heading Alveolar Abscess, may be here referred to as a localized form of suppurative inflammation in which the mucous membrane of the gums and cheeks becomes involved. Usually commencing from a carious fang, but by no means always so, the dental periosteum becomes inflamed and converted into an abscess-sac, which, increasing in size, destroys the thin alveolar wall of bone and bursts into the submucous tissue, in which it spreads, finally pointing on the surface of the mucous membrane. Accompanying the development of the abscess is a very extensive swelling of the face, which produces considerable distortion, particularly if the mischief be situated in the upper jaw. In most cases the boil bursts into the mouth in the groove between the gum and the cheek, or on the gum; sometimes, when the fangs of the teeth are long and extend beyond the level of the reflection of the mucous membrane on to the cheek,—seldom, therefore, occurring with the milk-teeth,—the abscess may burrow in the connective tissue and point on the cheek or under the chin; and very rarely, with inflammation at the roots of the upper incisors or canines, the pus may burrow and finally burst at the posterior margin of the hard palate.

Treatment.—If the tooth be too far damaged to be restored, it is well to extract it at once, and at the same time open the gumboil in the mouth with a narrow bistoury: the free exit of the pus in both directions soon allows of cure. If the tooth be not removed, a fistula is apt to remain from the open abscess-cavity to the carious fang, with a chronic discharge of matter. Extraction should at once be performed if the abscess be pointing on the surface of the skin, as it is highly desirable to avoid a superficial opening. Even after the removal of the offending tooth the abscess-cavity may fail to close, and may become converted into a serous cyst which may refill again and again, requiring for its complete cure such treatment as will insure its closure by adhesive inflammation. Hot fomentations or poultices to the face, unless the abscess be threatening to burst through the skin, and bread or fig poultices in the mouth, may be usefully employed to promote the suppuration and relieve the pain, which rapidly subsides with the swelling when the pus is evacuated.

SYMPTOMATIC AFFECTIONS OF THE MOUTH.

Under this title may be conveniently grouped those morbid conditions of the mouth which form but a part of various maladies and are not of themselves the primary or essential disease. All varieties of stomatitis are met with under these circumstances, from the simplest catarrh to extensive and characteristic ulceration; the mucous membrane of the mouth may also, like the skin, afford a site for certain eruptions, and there are a few other abnormal symptoms which may be described. Most of them will receive detailed reference in their appropriate places in this work, and are merely mentioned here for the purpose of completeness of this article.

In many of the acute specific fevers the buccal mucous membrane exhibits a more or less marked inflammatory state, or there may even be eruptions of the rash peculiar to the disease. As a cause of simple stomatitis measles has already been mentioned. The appearances may be little more than a mottled redness of the palate and inner surface of the cheeks, with an increase in the secretions; the blush on the soft palate, dotted with numerous minute red papules, may precede the cutaneous rash, or there may be very distinct swelling of the gums, and general hyperæmia of the mucous membrane, with here and there thin, pearly-white patches of proliferated epithelium, which gradually come off as the disease subsides; sometimes superficial erosions are to be noticed and aphthæ appear. Cancrum oris does not develop until after the measles has passed. In scarlet fever a moderate increase in the redness of the mouth is sometimes to be seen extending forward from the inflamed fauces. The characteristic vesicular and pustular eruptions of varicella and variola are occasionally seen on the inner surface of the cheeks, on the tip and edges of the tongue, and on the palate, where they may leave small ulcers. Diphtheria, as causing the formation of membranous patches in the mouth, has been previously referred to.

Syphilis is the cause of numerous affections of the mouth, though rarely if ever are they limited to this region. The oral mucous membrane may participate in the general swollen catarrhal condition so noticeable in the nasal chambers in congenital cases of the disease; more often are there to be seen mucous tubercles, fissures, or ulcers about the angles of the mouth or on the inner surface of the cheeks or on the palate, resisting all treatment except that of a specific character, and even with that sometimes proving very obstinate. The cicatrices by which the ulcers round the mouth are healed may so pucker and contract as very considerably to diminish the size of the oral aperture. The characteristic ulcer of the acquired disease has been met with in the mouth in children.

The affection of the mouth which forms one of the marked features of scurvy is essentially a stomatitis,—or, rather, gingivitis, since the gums are almost solely involved. There is a swollen, spongy condition of the mucous membrane of the alveolar border, which easily bleeds and is in older children associated with much fetor, especially when the disease proceeds to ulceration, as is very commonly the case. The livid gums separate away from the teeth, which in extreme cases fall out, and the inner surface of the cheeks and lips and the tongue are spotted with ecchymoses, some of which may become bleeding ulcers. It is, however, but one symptom of a general disease, which is treated of elsewhere, and which is not strictly a local affection of the mouth, nor, indeed, is it of so frequent occurrence in children as in adults. In those badly-nourished, ill-cared-for young children in whom the scurvy appears to be associated with rickets, as described by Dr. Barlow in this volume, there occurs very frequently, though not invariably, "a sponginess of the gums, with tendency to bleed, and some putrid odor." The swelling is most marked where the teeth are cut, and occasionally in place of the swelling "small localized ecchymoses have been seen beneath the gum in the situation of the on-coming teeth."[1]

Those profound conditions of malnutrition which favor rupture of the capillaries and escape of blood into the tissues lead to the production of petechiæ in the oral mucous membrane, such as are seen in the various forms of purpura.

The characteristic vesicles of herpes and the bullæ of pemphigus are not infrequently seen in the mouth, often leaving abrasions and ulcerations of the mucous membrane: more rarely are the eruptions of the varieties of urticaria met with in that situation.

In 1873 the late Dr. John Murray communicated to the Royal Medico-Chirurgical Society of London, in a paper afterwards published in the Transactions for that year, a description of a very remarkable condition affecting nearly equally three children in one family. Associated with numerous connective-tissue tumors of the scalp, forehead, neck, trunk, and extremities and a very peculiar condition of clubbing of the fingers was a peculiar state of the gums, which were "everywhere greatly hypertrophied and almost completely burying the teeth, forming in parts numerous papillomatous or polypoid-looking growths, and in other situations presenting a peculiar fungating appearance. The enlargement of the gums was most marked at their upper and free surface, where they were most flattened out and in parts hardened by the pressure of the opposing gums. They presented the natural color, and, although in parts somewhat soft, vascular, and spongy-looking, they mostly felt firm and fibrous to the touch. It was alleged that they had a tendency to bleed. The mucous membrane of the oral cavity was elsewhere in every respect normal, the disease being distinctly limited to the gums." This curious appearance was first observed when the teeth were cut, and some time before the tumors and the state of the fingers were noticed.

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[1] See Dr. Barlow's paper, Trans. Royal Med.-Chir. Soc., 1883.

Very similar cases, so far as the gums were concerned, are recorded by Prof. Gross,[1] Mr. Pollock,[2] Mr. Heath,[3] Dr. Waterman,[4] and Mr. MacGillivray.[5] In none of these children were there any of the fibromata and fibrous-tissue developments so marked in Dr. Murray's cases, although some were unusually pilose and most were of weakened intellect. Several of these cases are described as congenital, while in others the hypertrophy of the gums was distinctly stated as not noticed until the teeth appeared. Sections of the tissue when removed and submitted to microscopic examination disclosed nothing beyond overgrowth of the normal constituents of the mucous membrane. Paring off the hypertrophied gums was found to be of no avail, as the growth had invaded the alveoli and a recurrence of the conditions soon took place. Nothing short of complete removal of the bone and teeth with the gums has been effective.

Hemorrhage from the mouth, except when due to some local condition, such as ulceration, is rare, and in this respect contrasts with epistaxis. Along with bleeding from other surfaces it is a symptom of hæmophilia, and of a peculiar form of this disease described by Drs. Ritter and Epstein in the *Oesterreich. Jahrb. f. Päd.*, 1871 and 1876. This affection is strictly limited to the new-born, and it diminishes in severity with every week of life. A case of this rare condition is described in the *Archives of Pediatrics*, June, 1888, where a nine-days-old infant bled to death from the mouth after its tongue had been rubbed with a cloth for the cure of thrush.

Salivation is an event of frequent occurrence in children. Apart from the increased flow which usually accompanies dentition and the ptyalism caused by such drugs as mercury, the iodides, or pilocarpine, it is a characteristic symptom of several of the oral affections already described, such as ulcerative stomatitis and noma. Diseases of the gastro-intestinal tract and of the pancreas are sometimes complicated with this condition, possibly induced reflexly *via* branches of the vagus as the afferent nerve to the medulla and thence to the vessels and secreting structures of the glands. In rare cases of disease, tumors, etc., of the medulla oblongata or of the facial nerve, salivation has also been known to occur. A frequent dribbling from the mouth by no means necessarily implies increased secretion, since there may be failure in swallowing what is really but a normal quantity. Some very interesting cases of ptyalism have been seen in perfectly healthy children quite free from constitutional or local disease, and at ages from two to eight years, outside the period of dentition. Dr. Bohn,[6] who has specially called attention to these cases, affirms that the secretion is mostly excessive when the children are up and about, is much reduced when they lie down, and ceases when they sleep. He notes its emotional aspect, and regards it as a neurosis. The affection tends to disappear as the children grow up. The saliva when thus abnormally abundant is diffluent, with flakes containing epithelial cells and mucous corpuscles; it is apt to be mixed with viscid mucus when there is much stomatitis. It is alkaline or neutral, rarely if ever acid unless from decomposition of sugar, etc., in the mouth; the proportion of ptyalin is deficient.

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[1] System of Surgery, 1862.

[2] Holmes's System of Surgery.

[3] On Diseases of the Jaws, 1884, and in Trans. of Odont. Soc., 1879.

[4] Boston Med. and Surg. Jour., 1869.

[5] Australian Med. Jour., 1871.

[6] Gerhardt's Hand-Book of Diseases of Children, vol. iv., 1880.

For the salivation of the various forms of stomatitis chlorate of potassium is the surest remedy, whilst for the neurotic form iron or arsenic has been found most useful, notwithstanding that anæmia could not be assigned as the reason for the symptom. Atropine has a markedly controlling effect over the secretion, and when the cause is absolutely irremovable is a valuable palliative. Dr. Finlayson[1] records a case of idiopathic salivation in a strong, healthy child six years old who had formerly suffered from measles and pertussis, in whom the average amount of secretion was thirty ounces in twenty-four hours, and who was cured in a month by one-fourth grain of extract of belladonna given three times daily. This case was strangely complicated by attacks of severe abdominal pain lasting for half an hour, of entirely unknown cause.

The opposite condition of "dry mouth" (aptyalism or xerostomia of Hutchinson), due to suppression of the salivary and buccal secretions, has not been met with as an idiopathic affection in children, so far as I am aware. Occurring in adults as a temporary result of certain mental phases, as fright, or more permanently from unknown causes, it has only hitherto been noted in children as a symptom of the febrile state, or as the result of blockage of the nares, whereby the air to the lungs was made to pass through the mouth.

DISEASES OF THE TONGUE.

The tongue largely participates in the affections of the mouth which have been described, but there are several morbid conditions restricted in great measure to the organ which require description. It is also usual to regard the appearance of the tongue as in some degree indicative of the state of the stomach, and, although it is easily possible for many mistakes to be made by relying too completely on the association, nevertheless the characters of the tongue, as regards its shape, size, and condition of surface, are frequently representative of changes in other parts of the alimentary tract: these characters are for the most part referred to under their appropriate headings.

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[1] Glasgow Medical Journal, August, 1882.

ACUTE INFLAMMATION OF THE TONGUE.

This condition is occasionally met with in children, though perhaps with less frequency than in adults. It is essentially an acute affection, most commonly determined by exposure to cold and damp, very much as a quinsy is produced, and far less often by septic matters, corrosive substances, boiling fluids, injury, or the stings of animals which have gained entrance to the mouth.

Within a very few hours the disease is fully established, though with much variation in the degree to which the symptoms are developed. The most striking sign is the enormous swelling which the tongue undergoes, filling up, as it may, the cavity of the mouth and protruding beyond the lips, becoming at the same time excessively tender and painful, and coated with a thick fur except at the exposed parts, which are dry and cracked. There is a copious discharge of saliva. The obstruction caused by the swollen organ renders the patient unable to speak, and able only with difficulty to swallow or even to breathe. There is always a rise of temperature, though seldom above 101° or 102° F. Some swelling of the sublingual glands exists.

The condition tends to subside, and, as a rule, begins to do so within a few days, the tongue quickly regaining its normal size, frequently leaving some superficial sloughing and ulceration on the dorsum. The other symptoms rapidly diminish with the improvement in the tongue. Sometimes, whether from the virulence of the cause or from the idiosyncrasy of the patient, there follows a localized suppuration in the tongue-substance,—an abscess,—which gives rise to a variable-sized circumscribed swelling in one-half of the organ, which lasts perhaps for some time after the acute inflammation has passed off, and which, being unaccompanied by fever, may at first escape notice. Indeed, most of the cases (and they are not numerous) of abscess of the tongue which have been recorded have given no history of symptoms preceding the swelling or perhaps until after its rupture and the discharge of its contents, the abscess frequently running its course almost unnoticed by the patient, and when attention is called to it there may be no recognizable fluctuation and nothing beyond the swelling to be discovered.

Except for the obstruction, which may sometimes assume alarming proportions, acute glossitis is not a serious disease, and only in extreme cases calls for special treatment beyond a saline aperient and confinement of the child in a steamed atmosphere, giving such fluid food as can be most easily swallowed, and, if possible, ice to suck, whilst keeping the extruded part moist with glycerin and borax. Should active interference be required, leeches may be applied under the jaw, or an incision three-fourths of an inch to one inch long and one-fourth of an inch deep with a sharp bistoury into the substance of the tongue about one-half inch each side of the raphé affords the most effective and certain relief, the swelling quickly yielding to the moderate bleeding which ensues. If an abscess be diagnosed, or if a localized swelling have existed in the tongue for some time, with no enlargement of the neighboring lymphatics or ulceration of the surface over it, it is best to open by incision, with or without a previous exploratory puncture.

ULCERATION OF THE TONGUE.

The exposed situation of the tongue to irritation from ingesta, teeth, etc., determines the frequent occurrence of ulceration; and the same circumstance may account for its being a favorite locality for the manifestation of ulcerative destruction predisposed to by certain morbid constitutional states. The various causes which lead to the ulceration of the mouth for the most part tend to affect the tongue in a similar manner, and need not be referred to again beyond mentioning that while the disease especially known as ulcerative stomatitis is more generally limited to the gums and adjacent cheek and less often extends to the edges of the tongue, the simple form of ulceration, as from a broken tooth or from too irritating or too hot food, more frequently attacks the tongue than the other parts of the mucous surface. The tongue is affected by aphthous ulceration about equally with the rest of the mouth, and the ulcers which may follow herpes or the eruption of variola and varicella are more commonly seen on this organ than elsewhere in the mouth. Mercurial ulceration is extremely infrequent in this situation in children.

The primary ulcer of syphilis has been seen in children on the tip of the tongue, and fissures and cracks as well as ulcers of all degrees of depth are not uncommon as the result of the congenital affection; along the edge where the teeth may be the exciting cause, mucous tubercles are more commonly developed.

The tubercular or strumous ulcer is of very doubtful occurrence in children: I have met with no recorded case.

In a large proportion of cases of whooping-cough it has long been known that shallow oval ulcers with clean-cut edges and often covered with a yellowish layer are to be seen close to the frænum linguæ, generally single and very rarely double. They were formerly regarded as a primary phenomenon of the general disease, and were even compared to the initial sore of syphilis: it is now recognized that they are caused by chafing of the under surface of the tongue against the sharp lower incisors during the paroxysm of the cough. They are very difficult to cure so long as the cough lasts, but when that ceases they readily heal: they appear sometimes to be benefited by being touched with glycerole of tannic acid.

NEW GROWTHS OF THE TONGUE.

The tongue is sometimes even in children the seat of new growths. These are frequently congenital in origin, though they may not be noticed until some time after birth. Among others, rare cases have been recorded from time to time of papillomata or warty growths,[1] of fibrous-tissue tumors,[2] of fibro-cellular tumors,[3] of glandular tumors,[4] of sarcoma,[5] of keloid,[6] and of cysts.[7] Vascular tumors or nævi, at least the venous variety, are somewhat more common; they also are usually congenital, and form livid elevations on the mucous membrane of the anterior part of the tongue, giving rise to no pain or other symptom, except bleeding should they be wounded. They may increase in size, or shrivel, or undergo warty degeneration: for a well-marked case of the latter change see "Transactions of the Pathological Society," 1875. Syphilitic gummata have been found in the tongue in children.[8]

The appearance of the tongue in children is very variable, although probably not to the same extent as in adults, but the diagnostic value of these appearances is certainly very slight and rarely to be taken as evidence of the condition of the stomach. Mr. Butlin [9] showed that the fur on the tongue consists mainly of masses of organisms—mostly micrococcus and bacillus subtilis—adhering to the filiform papillæ, "the epithelium and food-débris being unimportant and, as it were, accidental constituents." The tongue in most children is slightly furred, and in infants this is attributable, among other reasons, to a deficient ability on their part to clean it. In some diseases this thin coating becomes thicker, as in scarlet fever, when the fungiform papillæ stand out large, red, and prominent amid a dense layer of white or dirty-white or faintly-yellowish fur; on the other hand, a severe gastro-enteritis may exist without the tongue undergoing any noticeable alteration. In some febrile states the surface may be dry, brown, and cracked, the denuded parts being bright red. The peculiar condition known as "black tongue" or nigrities, due to black particles in or upon the epithelial scales which cover the filiform papillæ,[10] has not been seen in children, so far as I know.[11]

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[1] Mr. Bryant, British Medical Journal, 1863, vol. i.; Mr. Butlin, Diseases of the Tongue, p. 247.

[2] British Medical Journal, 1885, vol. ii. p. 1061.

[3] F. Mason, Path. Soc. Trans., 1863 and 1866.

[4] Dr. Hickman, Path. Soc. Trans., 1869; R. Parker, ib., 1881.

[5] Jacobi, Amer. Jour. of Obstetrics, 1877.

[6] Sedgwick, Trans. Path. Soc., 1861.

[7] Dr. Hadden, Trans. Path. Soc., 1886.

[8] Dr. Barlow, Trans. Path. Soc., 1880. The same physician has told me of another case in a child, which ulcerated through to the surface.

[9] St. Bartholomew's Hospital Reports, 1879.

[10] Hutchinson, Med. Press and Circ., 1883, vol. xi.

[11] The normal occurrence of micro-organisms in the mouth is a subject of great interest. Vignal (Arch. de Physiol. Norm. et Path., 1886) has described and drawn a large number of different forms, including cocci, leptothrix buccalis, bacterium termo, bacillus subtilis, and many other bacilli and vibriones, as constantly occurring in the oral cavity, and, although under most circumstances their presence gives rise to no inconvenience or disease, it is at least open to question whether, under the conditions which an altered soil such as a state of fever (measles, etc.) may provide, some may not come to acquire virulent properties, the results of which are seen in noma and possibly in some forms of ulcerative stomatitis.

ERUPTIONS ON THE TONGUE.

The eruptions of variola, varicella, and herpes have been found on the tongue, the last with considerable frequency; and one case is on record where the tip of the tongue was affected with xeroderma pigmentosum.

A peculiar eruption limited to the tongue, described under various names, as the "wandering rash," "geographical tongue," "lichenoid" or "circinate eruption," was first referred to in France by Gubler.[1] An able paper on the subject appeared in the *Lancet,* May 10, 1884, by Dr. Colcott Fox, and Mr. Butlin in his work on "Diseases of the Tongue," 1885, describes it fully. The disease chiefly develops on the dorsum, and thence may extend over the edge to the under surface of the organ. It commences as one or more round, raised, whitish patches, which enlarge peripherally like "ringworm" (to which, however, it has no real resemblance), and very soon form a series of rings of heaped-up epithelium of a whitish or yellowish color, within which is a red zone of desquamated surface, whilst the centre is a red glazed area devoid of filiform papillæ, though the fungiform remain. As the eruption spreads, the centre areas become re-covered with epithelium, the duration of the rings being about six days, as stated by Parrot. The circles, meeting one another, form sinuous lines over the tongue, subsiding and advancing, perhaps for months or years. It is sometimes accompanied by much itching and salivation, but, except for associated dyspeptic troubles, there are usually no symptoms. The original idea that it was of a parasitic nature has been quite discarded, since no special fungus has been found in connection with the rash, and there is no evidence of any relation existing between it and syphilis, as was described by Parrot; some regard it as a tropho-neurosis. It has been seen in infants a few months old, and the majority of cases occur under the age of two years; exceptionally adults and old people have suffered. The sexes are equally affected. It has been known to recur. From its harmless nature, the malady calls for little treatment,—to which, indeed, it is very resistant; scarcely anything, local or general, can be said to influence much the course of the disease, which appears to wear itself out. Slightly astringent or soothing washes and tonics may be tried.

RANULA.

The condition to which this term is applied consists, in the great majority of cases, of a cystic distention of the Blandin-Nuhn (mucous) gland, situated on the floor of the mouth immediately under the tongue. It undoubtedly occurs, though rarely, as a congenital affection,[2] and subsequently at any time of life. The determining cause of the obstruction of the acini of the gland is probably inflammation induced by various irritants to which the tip of the tongue is exposed. Another view of the pathogeny of the condition is held by Suzanne,[1] who has made careful dissections of the cyst; according to him, it is a mucous transformation of certain acini of the sublingual gland, and the mucous glands are not the seat of the disease. Microscopically the cyst-wall is lined by columnar epithelium in various stages of proliferation and mucoid degeneration, the swelling itself being bounded by a fibro-elastic layer over which is stretched the thinned mucous membrane. The size of the tumor thus produced varies from that of a pea to that of a walnut, and it is to the size alone that any inconvenience or symptoms, such as interference with the movements of the tongue, are due. Usually limited to one side of the frænum linguæ, it protrudes as a tense, bluish, translucent swelling, over which several large vessels are to be seen; sometimes the cyst itself is bilocular, and very rarely there is a ranula on each side. The contents are mucus more or less viscid: the fact that they do not consist of saliva, coupled with the circumstance that Wharton's duct and the submaxillary gland are generally quite normal, has led to the overthrow of the older view that ranula was due to obstruction of the salivary duct. But Suzanne's researches show that the affection may sometimes really be a mucoid degeneration of some lobules of the gland, leading to an accumulation of mucus, and not of saliva, as would be the case in a simple retention-cyst.

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[1] Dict. encyc. des Sci. méd., article "Bouche," 1869.

[2] Lond. Med. Record, 1877, p. 497, various references.

For the successful treatment of this condition extreme measures should be adopted at once. Merely emptying the sac by puncture is insufficient; it will refill again and again: adhesive inflammation should be set up by a silver-wire seton, or by removal of a part of the cyst-wall, leaving the rest to heal by granulation. Some surgeons recommend complete dissection out of the cyst and any portion of gland which may remain, while others are content with injecting the cavity with a few drops of a one-in-ten solution of chloride of zinc or touching it with a galvano-cautery. But, although the submaxillary gland and its duct take no part in the production of what is ordinarily known as ranula, the latter may be obstructed by a salivary calculus, leading to great swelling and tenderness of the gland itself as well as of the tongue and subjacent parts of the floor of the mouth. Two interesting cases are detailed by Dr. S. Mackenzie.[2] The nature of the cause being detected by a probe passed into the duct, removal of the calculus by incision should be at once performed, when the symptoms soon subside.

DERMOID CYSTS.

The floor of the mouth is also the seat of tumors of another character, which are often called sebaceous, from the soft cheesy nature of their contents; more properly considered, they are dermoid cysts developed from an infolding of the superficial layer of the blastoderm. Doubtless congenital in origin, it may not be until adult life that they are noticed, and relatively few, therefore, are met with in children. The swelling they give rise to is usually confined to one side, of a yellowish color, and not translucent like ranula, and sometimes, extending deeply between the muscles, may be perceptible externally between the chin and the hyoid bone. The contents are often offensive in odor and of varying consistence, being composed of fatty matter, epithelial débris, and cholesterine crystals, sometimes also containing a few hairs, some of which are attached to the inner surface, which closely corresponds in structure to the skin. Complete removal of the unopened sac, if possible through the mouth, is the most satisfactory treatment. Merely emptying it by incision leaves a wound which is very intractable in healing.

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[1] Arch. de Physiol. Norm. et Path., Series iii. vol. x.

[2] Practitioner, 1881, vol. xxvii. p. 266.

HARE-LIP AND CLEFT PALATE.

BY J. FORD THOMPSON, M.D.

HARE-LIP.

HARE-LIP is one of the commonest of congenital deformities. It bears a striking resemblance to the natural cleft in the lip of the animal from which it receives its name, but differs from it in being almost always to the side of the middle line of the upper lip, and not in it, as is the case in the hare. It consists in a vertical fissure or fissures through a part or the whole of the lip, the result of arrest of development in early embryonic life.

A knowledge of the process of development of the face renders it easy to comprehend all the congenital defects of the mouth, but it will be necessary here only to call attention very briefly to the part in which we are immediately interested.

The central process, or fronto-nasal plate, descends from the cranium in the middle line in front, and from it are formed the prominent portion of the nose, septum nasi, columna, intermaxillary bone, and the middle part of the upper lip. On each side there descends a lateral process, the superior maxillary plates, from which are developed the cheeks, the superior maxilla, and the sides of the upper lip. In normal development these lateral centres unite posteriorly to form the soft and hard palates, and in front they fuse with the central process to complete the alveolus and the upper lip, the lines of union in the soft parts being beneath the nostrils.

The lower lip is formed from the inferior maxillary centres, which meet in the middle line and join the superior maxillary centres on each side in a line extending from the angles of the mouth. The union of these different segments is complete about the tenth week of fœtal life.

Arrest of development is very rare except between the central and the lateral processes, but why these should be so much more frequent is not apparent, although it seems probable that their more tardy development may in some measure account for it, as it is well known that such defects are more likely to affect parts and tissues of slowest growth.

As to the determining cause of the arrest, from the present stand-point, absolutely nothing is known. It is probable that very few professional men at present attach any importance to the explanations that mothers are ever ready to give of some nervous shock or maternal impression experienced during their pregnancies; for upon investigation it will almost always be found that these sights have been seen long after the period at which they could possibly have influenced the development of the parts in question. It is seen, however, at a glance that non-unions between these centres will give rise to congenital clefts, and the various grades, varieties, and complications of hare-lip and cleft palate are made intelligible.

The defect may be limited to the soft parts upon one side of the central portion of the lip, or it may affect both; it may extend backward and inward through the alveolar border towards the middle line upon one or both sides of the incisive bone, as the cleft of the soft parts may be single or double; and it may extend through a part or the whole of the hard and soft palates.

Males are more frequently affected with hare-lip than females; and it sometimes happens that the child is afflicted with one or more other congenital defects, or that such conditions have been observed in other members of the family; showing, as is generally admitted, that heredity is a determining influence in a certain percentage of cases.

Simple Hare-lip.—Simple hare-lip is a fissure involving more or less of the height of the upper lip, situated on either side of the median line, but more frequently to the left. It may be complete (Fig. 1) or incomplete (Fig. 2). It is sometimes so slight as to cause a mere indentation or notch in the free border of the lip, and from this there are gradations up to complete severance of the two sides, with the fissure extending into the nostril. All cases falling short of the nostril are classed with the incomplete variety.

Fig. 1.

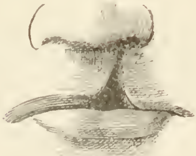
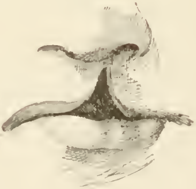


Fig. 2.



The margins of the cleft are covered with mucous membrane, and are in every respect like the free border of the lip, with which they are continuous. The gap increases in width from above downward by the action of the muscles, and from the same cause is exaggerated in crying or laughing. The inner side is more nearly vertical, the other being drawn outward and frequently much rounded off below, so as to present an obtuse angle at its juncture with the free border; the sides are often of unequal size and length, especially in the complete variety.

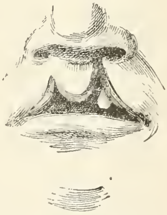
In the imperfect cases there is rarely any additional deformity. Functional disturbance is comparatively slight, although at first there may be some difficulty in nursing, and later in life, of course, if not remedied by an operation, pronunciation will be imperfect. When the fissure extends into the nostril the deformity is usually much greater: the nose is flattened and the nostril of the affected side widened by the drawing outward of the ala by the action of the facial muscles; nursing may be somewhat more embarrassed, but rarely sufficiently so to interfere materially with proper nourishment.

There is a form of simple hare-lip occasionally observed in which a deep groove, more or less wide and long, occupies the usual position of the fissure. In these cases the arrest of development has affected only the muscular tissues of the lip, the skin and mucous membrane being continuous from side to side.

A few cases of central fissure have been reported, but they are too rare to require any special description. They are clearly the result of non-union between the two parts of which the median tubercle is at first composed.

Double Hare-lip.—Uncomplicated double hare-lip is characterized by the presence of two fissures, and an intermediate portion, the lunula (Fig. 3). Both fissures may be complete or incomplete, or, as more often happens, one side may extend to the nostril, usually the left, while the other affects only a part of the lip. When both sides are partial the deformity is confined to the lip, but when one or both sides are complete there is a corresponding deformity of the nose, the tip being flattened and the nostrils expanded. The central portion is of variable shape and size, most frequently triangular, and scarcely ever so long as the lateral portions. The gap below the median tubercle is much wider than in simple cases. Suckling is sometimes practicable, but spoon-feeding has to be resorted to in many cases.

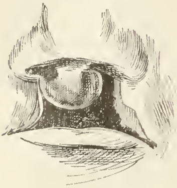
FIG. 3.



Complications.—Both single and double hare-lip, more frequently the latter, may be complicated by a more or less extensive fissure of the palate. When the defect in the lip is unilateral that of the bone is likewise so. The gap in the soft tissue is usually wide, and the two sides of the jaw are often upon a different level, with prominence of the incisive bone in the middle line.

Bilateral hare-lip may be complicated by double or single fissures of the jaw, which sometimes extend only the distance of the intermaxilla, in other cases implicating a part or the whole of the hard and soft palates. The appearance of the alveolar arch when there is but one cleft is the same as that just described, but when there is one upon each side of the incisive bone there is, as a rule, much greater deformity. There is quite constantly marked projection forward of the central portion, with a wide cleft upon either side of it, separation of the two sides of the lip to an extreme degree, with flattening of the nose and expansion of both nostrils (Fig. 4). This displacement is sometimes so great as to be attached to the septum so high that it appears to hang from the end of the nose, the piece of integument covering its upper surface being nearly in line with the dorsum of the organ.

FIG. 4.



It is to be noted that, notwithstanding the generally accepted theory that the arrest of development is in the line of normal union, the incisive bone in these cases does not always contain the four incisors, nor is the cleft constantly between the second of these teeth and the canine tooth, but it is quite often to the outer side of the first incisor.

It is possible, perhaps, that the undeveloped condition of the bone itself will account for some of these differences, or that there are, as contended by some authorities, more than two original centres for this intermediate portion.

The condition of the infant in complicated hare-lip is much more grave than in the previous varieties, and requires the most careful nursing to carry it safely through the first months of life. The impossibility, often, to nurse, the difficulty in deglutition, with regurgitation through the nose, and the constant irritation kept up by such conditions, cause the death, unquestionably, of a large proportion of these cases in early infancy. Indeed, in simple cases there appears to be a predisposition to intestinal and respiratory diseases, and as far as statistics go they show a larger mortality in children thus afflicted than in those of normal conditions.

Treatment.—Before describing the various operations for hare-lip, it is well to consider briefly some of the questions of interest concerning certain points about which there is a lack of unanimity on the part of surgeons.

There is considerable difference of opinion, for instance, as to the most favorable age of the patient. Many prefer (especially is this the case with the German surgeons) a very early period—during the first few weeks, or even the first few days—for both simple and complicated cases; but the majority prefer deferring all operative procedures till the child is somewhat developed, and better able to stand the nervous shock or the necessary loss of blood. It is true, when the deformity is slight immediate operations may often give very satisfactory results, and when there are reasons for such early interference it may be done; but, as even a trace of the deformity becomes in after-life a source of mortification to the patient and the family, it is better to operate under the most favorable conditions and with all the care possible, in order to make the lip as nearly natural in appearance as practicable. It is not necessary or desirable, however, to postpone interference, even in bad cases, so long as is recommended by some authorities,—*i.e.*, till after dentition. It may be said, in a general way, that for ordinary cases the age between six weeks and three months is to be preferred, the exact time depending upon the condition of the patient as regards health.

In bad cases of double hare-lip, and in all cases of complications in which it is necessary to deal with projecting intermaxillæ, or to dissect up the lip and cheeks in order to free the alæ of the nose and to bring the parts together without tension, it is well to defer the operation three or four months longer, when, the child being in good condition, it may be performed with reasonable prospect of success. There is a very great advantage in operating comparatively early when cleft palate is a complication, as the constant pressure of the united lip upon the as yet soft bone soon closes the gap in the alveolus; no thought of treatment of the fissured palate beyond the restoration of displaced intermaxillæ should be entertained at this time. Occasionally cases are seen in which, on account of excessive deformity and enfeebled condition, it will be wiser to follow the advice of those who advocate waiting till after dentition.

It seems impossible to give any satisfactory statistics as to the mortality of the operation itself. Fritsche estimates it at two per cent. in the first two weeks, and as high as fifteen per cent. from this age up to three months. This is probably above the average; but the dangers in complicated cases are certainly to be seriously considered.

The position of the child for operation depends upon whether an anæsthetic is to be used or not. When the child is only a week or two old, and the case is a simple one, anæsthesia is certainly unnecessary, as the operation is performed rapidly and without much suffering. The infant should be held upright in the lap of a nurse, securely wrapped in a towel or sheet. An assistant stands behind the nurse's chair to steady the child's head with his hands and at the same time to support the cheeks. The operator sits facing the patient, with an assistant at his side. This position is much to be preferred to that with the child's head somewhat dependent between the operator's knees, which allows the blood to drop into the mouth and be swallowed, besides the graver objection that it seems impossible in this reversed position to trim and adjust the parts with the nicety and exactness required.

When the child is a little older it should be placed upon a table in a good light, and chloroform administered by dropping a few drops on a handkerchief held over the mouth and nose. But a very small quantity is required to anæsthetize the patient sufficiently, and when carefully given is without danger. In these cases chloroform is in every way more satisfactory than ether. The operator stands or sits to the left of the table, with the child's head turned towards him.

But few instruments are needed. The older surgeons used scissors for freshening the borders of the fissure, and some do so at the present day; but it is generally admitted that the knife is preferable, as it is almost impossible to use the scissors to advantage except in the simplest class of cases, and then they possess no superiority. A very sharp, narrow-bladed knife is the best: Von Graefe's cataract-knife answers admirably. There is no need of a wooden spatula or support under the lip to cut upon, as it is in the way and useless.

Hemorrhage is controlled by the assistant grasping each side of the lip as it is cut; or Smith's clamp-forceps may be used; or, in default of these, a couple of ring-forceps with a narrow piece of rubber tubing slipped over the handle for making pressure, as described by Erichsen, will be found satisfactory. A rat-tooth forceps is used for holding the angles during the paring or in forming flaps, or the angles may be very nicely manipulated by passing a loop of silk through them as the first step of the operation.

Until quite recently the twisted or hare-lip suture was almost universally used, as it was thought to possess special advantages; but of late years, here as elsewhere, it has fallen somewhat into disrepute, whether justly or not appears yet to be an open question. The majority, probably, of modern surgeons have discarded it, and rely upon the simple interrupted suture of various materials.

When the flaps can be brought together without tension the simple suture is all that is needed, but care must be taken not to draw the parts too closely together,—a common error with the inexperienced,—or there may be an infolding of the anterior margins of the wound, which will interfere with the proper union of the parts. The common objections to the pins are that they leave scars, are less cleanly than sutures, and prevent the nice application of supporting strips of adhesive plaster.

As to the first objection, it is questionable whether it is well taken, for when the pins are of proper size, and withdrawn between the second and the third day, as they should be, there certainly is left no more disfigurement than from other sutures, especially those of silk. In fact, this scarring does not occur after any of the sutures recommended except when they are left in too long: they should be withdrawn, almost without exception, by the end of the third day. There is some weight in the other objections, but they are not of a serious nature, as they are readily overcome by a little care and attention. It would seem in many cases that a combination of the two kinds of suture might give the best results: a pin for the lower part of the wound, and interrupted sutures above.

In cases of much tension the supporting or relaxation suture renders excellent service; only one is needed, the wound proper being closed by interrupted sutures. Two small buttons of lead, perforated in the centre, with a couple of perforated shot and a piece of small silver wire, constitute this suture. A long slender needle is used for carrying the wire, which is inserted about an inch from the margin in the lower portion of the lip and passed through the tissues to the mucous membrane, but not through it; it is then reinserted and brought out at the same relative position on the opposite side. The buttons and shot are then slipped over the wire of each side, or one end may be prepared beforehand, the parts drawn together sufficiently to relieve all tension, and the shot clamped. With this suture all other support may be dispensed with, though it is not in the way of the usual application of adhesive strips.

Of the materials used for the interrupted suture the choice lies between silver wire, silkworm-gut, and antiseptically prepared Chinese twist silk, all of which are good, with a slight advantage, perhaps, in favor of the first-named. Horse-hair is unreliable, at least for the principal sutures, and catgut possesses no qualities which would recommend it in this operation.

Antisepsis should be observed as closely as is practicable, all the usual precautions, now too familiar to need mentioning in detail, being taken to prevent contamination of the wound, although it is impossible to apply a perfectly antiseptic after-dressing in these cases, which, however, appears less to be regretted than in wounds generally, as it very rarely happens that healing is interrupted or prevented by those surgical accidents which are to be attributed in general to defects in this particular.

What is now known as the old operation for hare-lip is exceedingly simple and easy of execution, consisting merely in cutting away the rounded edges of the cleft, bringing the two plane surfaces together with sutures, and retaining them in apposition until union by the first intention shall have taken place; in addition to this, the frænum is cut, as well as any abnormal adhesions of the lip to the gum which may prevent easy coaptation. The cosmetic results obtained by this method are rarely creditable to the operator, for it seldom happens that the two sides of the lip are of so nearly the same length or shape as to be brought together without the ugly notch at the lower border resulting which is so often seen as a disfigurement. Even in cases which would appear to be remediable by this simple procedure, more or less retraction of the cicatrix is likely to follow the healing process, and thus produce the depression in the vermilion border.

It is better, then, in these cases to abandon the scissors altogether, and to proceed as follows, after the method of Malgaigne. The child being in position and everything in readiness, the operator seizes one of the lower angles with the forceps and puts the lip upon the stretch, while compression is made by the clamp or the fingers of the assistant; the lip is transfixed above the vermilion border, not too near the mucous membrane of the cleft, and the incision carried upward well above the superior angle. The opposite side is treated in the same way, thus forming two flaps with their bases downward. After freeing the lip sufficiently from the bone with knife or scissors, the flaps are turned down, and the raw surfaces united by

FIG. 5.

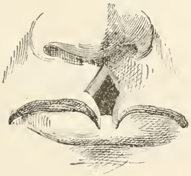
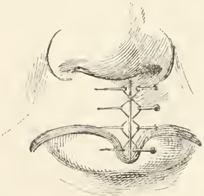


FIG. 6.



pins or sutures, or a combination of the two. Silk sutures are to be used for the flap and mucous border, and it is well to apply one or two to the inner side of the lip to bring the mucous membrane nicely together.

The needles for the principal sutures should be introduced about a fourth of an inch from the edge and carried through the lip to the mucous membrane and out upon the other side at the same distance. The one at the base of the flap should be inserted first, and in this, as in all other operations for hare-lip, the greatest care should be exercised, in approximating the lower borders, to avoid the "step" or break in the tegumentary line above the mucous border.

If the flaps be too redundant they may be retrenched by cutting through their bases from above downward and inward, thus leaving a less prominent protuberance than that shown in the figure; although it must be borne in mind that considerable shrinkage of the tubercle always follows in the course of a few months, and that then, if too prominent, it is easily remedied by a very simple operation.

After the parts have been cleansed and dried, the cheeks should be pressed forward, and a piece of rubber adhesive plaster, cut wide at the ends and narrow in the middle, should be applied from side to side to relieve traction upon the sutures, which it does, when nicely fitted, quite as well as the more expensive cheek-compressors and trusses recommended.

The child may be given the breast immediately after the operation is completed, as it assists in quieting it.

After forty-eight hours the plaster should be carefully removed by first detaching the ends and pulling them forward, while the cheeks are supported, and the wound examined. If it looks well it is perfectly safe to withdraw some of the sutures and then again apply the adhesive plaster. The next day all of them should be removed, and the treatment continued for a week or more by the plaster support alone.

If after removal of the sutures it should be found that union is imperfect, one or two pins may be introduced, with the hope of obtaining secondary union.

This plan of utilizing the parings for lengthening the lip is better than the curved and circular incisions practised by some surgeons for the same purpose, though these are an improvement upon the old straight cut.

When the fissure is shallow, Nélaton's method answers admirably, being, in fact, a modification of the one just given, but perhaps a little more simple. The lip is transfixed well above the border, and the incisions made around the upper angle and down the opposite side to a point on a level with that of entrance of the knife (Fig. 7). It will be seen that this differs from the above method only in leaving the flaps attached above,—or, rather, there is but one continuous flap, embracing both sides of the fissures, which when turned down present the lozenge-shaped wound shown in Fig. 8. When the freshened surfaces are united a prominence is formed below.

This method is also to be preferred for filling out the lower border of the lip when a notch has been left from a previous operation.

FIG. 7.

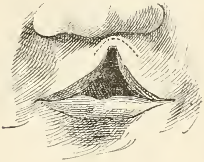
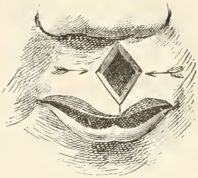


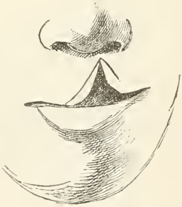
FIG. 8.



Single Flap (*Mirault*).—While the above operations very effectually prevent the ugly gap at the vermilion border, the objection has been urged against them that they also leave a deformity which persists or has to be remedied by further surgical interference. As this resulting protuberance is not in the middle line of the lip, it is more conspicuous, and to avoid this many prefer to practise the single-flap method, which certainly possesses the advantage claimed.

Upon one side, usually the shorter, a flap is formed, as in Malgaigne's plan, with its base left attached below; but upon the other side the mucous border with a strip of skin is cut from the fissure and the adjoining border of the lip in the line shown in Fig. 9. The obtuse angle of the freshened side is fitted into the angle above the flap, and the flap itself is stitched to the pared border of the lip.

FIG. 9.



The single-flap and double-flap methods, as detailed and illustrated above, usually give satisfactory results, but they may be sometimes advantageously modified both in the paring and in the formation of the flaps.

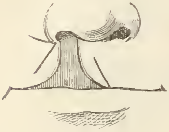
For instance, in freshening the upper angle by the common incision ∧, if the sides be of unequal length, when brought together there will result a puckering under the nostril, impossible to overcome; if, however, this angle be cut away in a horseshoe shape, nothing of the kind can occur. Instead of the usual flaps, an incision is made from the lower end of the circular incision outward and slightly downward through the lip upon each side.

In like manner the single-flap method may be modified in many cases with advantage.

These comparatively simple operations almost always give satisfactory results in simple hare-lip, if the paring be done with a free hand and the lip sufficiently freed from the alveolus; but when the fissure extends into the nostril with a wide gap between the sides, it is sometimes better to have recourse to one of the two following methods, both of which are well devised and when properly executed give admirable results.

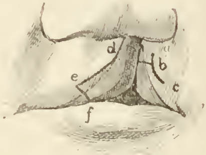
Giraldes's method is a double-flap operation, but differs from Malgaigne's in that the base of one of the flaps is left attached above beneath the nostril, whilst the other is made in the ordinary way (Fig. 10). After thoroughly freeing the outer side of the lip and making a horizontal incision outward under the ala of the same side, the superior flap is turned upward across the nostril, and the inferior downward to be united to the raw surface from which the other has been cut. This dovetailing of the flaps increases the depth of the lip, and gives a natural-looking floor to the nostril. A hare-lip pin, or, better, a relaxation suture, about the middle of the lip will render good service.

Fig. 10.



Colles's Operation.—This is also an excellent method for complete and wide clefts. One side, the shorter and more rounded, is freshened in the line *def*, Fig. 11; the other is transfixed at *a*, a little below the nostril and about an eighth of an inch from the margin, the incision made downward to *c*, thus leaving a flap attached at both ends, which is to be bisected at *b*, making one flap with its base above, and the other below. After freeing the lip and ala, the superior flap is turned upward with its cut extremity looking into the nostril, and its raw surface stitched to the upper part of the line *de;* the lower segment is turned down and stitched to the line *ef*.

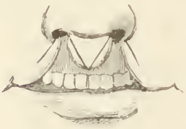
Fig. 11.



Operations for Double Hare-lip.—In uncomplicated cases of double hare-lip, when the central piece is on a level with the sides, the operation is scarcely more difficult than in simple cases, though somewhat more tedious.

The old plan of paring the margins by straight incisions will not answer at all, as a deep notch in the centre of the lip is inevitable after it. The double-flap method is to be preferred for the majority of cases, and the flaps are formed after the method of Malgaigne, as already described. The intervening piece of integument is pared to a V shape, as shown in Fig. 12, and loosened from the bone a little at the margins. The flaps are turned down, and the raw surfaces of the sides stitched to those of the median tubercle and below its apex to each other. A pin or supporting suture should be used below the middle of the lip, and the flaps nicely fitted together by small silk sutures. The supporting straps are applied as already described, or a Hainsby's truss may be substituted, if thought advisable.

Fig. 12.



Unfortunately, the majority of cases of double hare-lip are complicated with cleft palate, partial or complete, and very often with more or less displacement of the intermaxillary bone, which often render the operations much more prolonged and difficult. Even in the milder forms with unilateral cleft of the alveolus, the nose is flattened, and one or both nostrils drawn out towards the cheeks; here it is necessary to liberate the lip very freely from the bone, carrying the dissection up under the ala or alæ as the case may require, supplementing this, when not sufficient, by a curved incision outward through the lip around the wing. If the incisive bone be too prominent to admit of easy apposition of the soft parts, it should be grasped by forceps with rubber tubing over the blades, and forced into position by fracturing its attached side. It is unnecessary to apply sutures to the bone, as the pressure of the lip will keep the parts in contact or soon bring them together; but it is well, in cases in which the two sides may be easily made to touch, to freshen the apposed sides and unite them by superficial sutures to the gum.

It is to be remarked that this constant pressure of the united lip is capable in a few months of closing quite a gap in the alveolus, even when no attempt has been made to replace the projecting portion at the time of the operation.

When the cleft is double without undue prominence of the middle piece, or after it has been forced back into position, the modification of the double-flap method shown in Fig. 13, from Koenig, is advisable. After properly paring the lunula an incision is made upon each side a little above the middle, outward and a little downward, through the lip, from *a* to *b*. The part above *a* is not freshened, but pushed upward to form the floor of the nostril, whilst the line from *a* to *b* is stitched to the central tip, and the two flaps turned down and secured in the middle line.

FIG. 13.

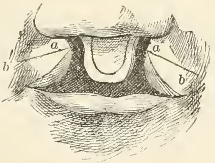
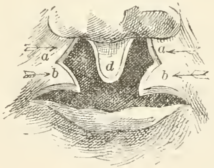
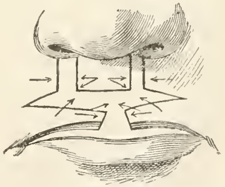


FIG. 14.



Occasionally the intermediate portion is of such size and shape that it may be utilized to greater advantage by trimming it to a ⊔ form rather than to a ∨; and in such cases the method of Koenig (Fig. 15) is particularly applicable.

FIG. 15.



After trimming the central tip square, the sides are freely freshened in their whole length, and then a lateral cut is made outward on each side, as in the above method, and the parts are united as shown in the figure. In this method it is essential that the soft parts be freely loosened from the bone.

In those cases of extreme displacement of the intermaxillary bone where it appears to hang from the upper portion of the septum or end of the nose, the operation becomes one of the most difficult and serious in the surgery of childhood, besides the almost impossibility of obtaining satisfactory cosmetic results. The gap between the lateral portions is very wide, and the skin over the displaced bone is almost on a line with the dorsum of the central tip of the nose, and the difficult question to determine is how to utilize it to best advantage in preventing or diminishing subsequent deformity of the nose and mouth.

The bone should never be sacrificed if there be a possibility of restoring it to its normal position; for, besides its usefulness in serving as a support for artificial teeth, its loss entails that ugly underhung appearance in an extreme degree, which is a sad deformity in itself, due to the necessary shortening of the alveolar border.

When the attempt to restore it is thought advisable, it is best done by Blandin's method of cutting out a wedge-shaped piece of the septum and then forcing the bone downward into position by breaking the remaining attachment. Very serious hemorrhage is liable to occur from the septum, which may require pressure, perchloride of iron, or, better, Paquelin's cautery, for its arrest.

Much has been written as to the advisability of using sutures to the bones to retain them in apposition during the healing process, and to this end, and also to control hemorrhage, Bruns devised the plan of passing two ligatures through the septum, one of which is tied before the wedge is removed, and the other secured over the intermaxillary, after it has been pressed back into the gap from which the V-shaped piece has been removed.

Langenbeck's modification of fixing the intermaxillary in position by dissecting from it and from the palate-process of the maxilla flaps composed of mucous membrane and periosteum and uniting them by sutures will also sometimes succeed, but, like the foregoing, much prolongs the operation, and thereby increases the danger.

When it is thought expedient to use either of these methods of securing the loose bone, it is better to divide the operation into two stages, leaving the lip to be dealt with after success or failure of the first. If the operation is to be completed at one sitting, it is only necessary to freshen the apposed surfaces and apply fine silk sutures to the mucous membrane, the pressure of the united lip assisting materially in retaining the piece *in situ*.

The development of the incisor teeth, which vary in number, is not necessarily arrested by these methods of replacing the bone, but it generally happens that they are so irregular and misplaced as to require extraction, the bone, however, serving the useful purposes already indicated.

But there are many cases in which the projection of the intermaxillary bone is so great that any attempt to preserve it would be worse than useless: although its sacrifice will be followed by shortening of the upper jaw, with consequent deformity of the face, it is the best that can be done under the circumstances.

The irregularly-shaped piece of skin extending from the end of the nose over the bone is to be dissected off from below upward, and left attached above to the nose to be utilized in the formation of the columna, the bone itself being cut away with a knife or bone-forceps, care being taken to control at once the hemorrhage from the vessels of the septum.

For the lip, in such cases, the methods of Malgaigne and Koenig are the best, but in addition the lip must be freely dissected from the maxilla, and incisions made outward through the lip and around the alæ in order to bring the parts together without tension. The lower end of the central tip left hanging to the nose is now carefully stitched to the upper angle of the wound, to form the columna: any defect in its appearance is to be left for subsequent treatment.

It is well to use in these complicated cases a supporting suture, and the ordinary sutures may be left in longer than has been advised for the simple varieties, as the slight additional disfigurement of puncture-scars is scarcely to be considered in operations of such magnitude.

CLEFT PALATE.

Cleft palate is a congenital fissure of the palate, most frequently limited to the velum, but quite often involving more or less of the hard palate as well. The defect may be so slight as to present a mere notch of the uvula, and from this there are gradations up to complete separation of the two sides of the palate and alveolus; in these latter cases the line of division extends forward in the middle line as far as the intermaxillary bone, and obliquely to one or both sides of that bone, to and generally through the lip, thus producing the complication of single or double hare-lip.

The hard palate alone is not nearly so often affected as the soft palate. When the fissure is complete through both palates, either the lower border of the vomer is attached to one of the palate-processes, or it hangs free in the nasal cavity, otherwise normal in appearance, or distorted and rolled upon itself to one or the other side.

The width of the gap is variable, and the arch of the palate is greater than natural.

The malformation is due to an arrest of development of the lateral or superior maxillary centres; but we are as ignorant of the determining cause in this as we are in other congenital deformities.

It appears that idiocy is not uncommon in those afflicted with cleft palate; and there is authority for the statement that impotency is frequent in males, whilst females escape this additional affliction. It is also noted that fissures of the palate are much more frequent in females, whereas hare-lip is more common among males.

The condition of the infant in the higher grades of fissure is a pitiable one, and calls for all the resources of the art of nursing to enable it to pass successfully through the first few weeks of its existence. The naso-buccal septum being absent, it is unable to suck, and even with the most careful feeding there is more or less regurgitation of fluids into the nose, with consequent distress and irritation, which, combined with imperfect deglutition, cause general impairment of health from insufficient nourishment. At a later period speech is much embarrassed, this embarrassment becoming, indeed, the principal inconvenience.

The mother's milk or that from a nurse should always be used for feeding the infant, but, as it cannot suckle or readily swallow from a spoon, some specially-devised appliance for at least partially overcoming the difficulty should be resorted to, such, for instance, as the flap of india-rubber attached to the nipple of the feeding-bottle, as advised by Mr. Coles, which is pressed against the gap in the effort to suck. After a few weeks of careful feeding many learn to swallow with comparative ease, and soon begin to take on flesh, though the mortality at this early age is very great.

With a view of improving deglutition, several surgeons have advised and practised very early closure of the cleft by operation ; but the results have not been such as to bring this practice into general favor ; and now few have any other purpose in the operation than favorably to influence the power of speech. To accomplish this comparatively early interference is necessary, before the habit of imperfect articulation, or talking through the nose, is acquired.

But few children begin to pronounce words with any distinctness before the end of the second year, and experience seems to have proved that from this age up to three or four years, besides the period being more favorable for surgical treatment, we may expect to obtain all the possible advantages of the operation, which, however, are not always entirely satisfactory.

Children, even after the most successful closure of the fissure, need most careful training and instruction in articulation to enable them to talk in a natural tone of voice.

In cases of cleft palate complicated with hare-lip the latter should be repaired at the age of three or four months, the pressure of the united lip acting quite effectively in diminishing the width of the gap in complete clefts.

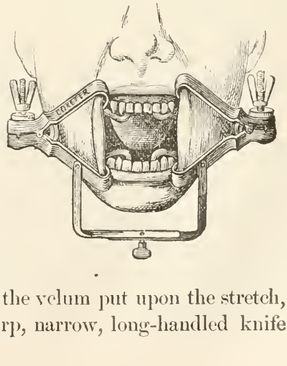
Before the use of anæsthetics was thought practicable, and before the invention of the combined gag and tongue-depressor, the operation upon the palate was postponed to an age when the intelligence of the patient might aid the surgeon in his manipulations ; but now there is no reason why it should not be undertaken at three or four years of age with expectation of procuring good immediate results, and with much better prospect of success as regards the effect upon the voice than at a more advanced period.

In cases of cleft involving both the soft and the hard palate it is still an open question whether it is better to attempt the complete closure of both at one sitting, or to operate first upon the velum and leave the more difficult part for subsequent treatment. It would seem to be advisable to adopt the latter plan, as the former greatly prolongs the operation and adds to its dangers; besides, the united velum has a tendency to diminish the width of the remaining cleft.

To Roux is due the credit of first popularizing the operation of staphylorraphy, but to Sir William Fergusson more than to any other surgeon are we indebted for certain improvements in the technique which have materially assisted in establishing it upon a scientific basis. Before his demonstration of the necessity of myotomy as a preliminary step or an essential part of the operation, the margins of the fissure were merely pared and brought together by sutures, with almost invariable failure to unite. He showed that this failure was due to the constant tugging of certain muscles upon the line of union, the principal one at fault being the levator palati, and the next in importance the palato-pharyngeus. His first step then was to cut the former muscle with a right-angular knife passed through the fissure, and an incision made perpendicular to the centre of a line from the hamular process to the orifice of the Eustachian tube. The palato-pharyngeus was then readily severed with scissors, after making the posterior pillars prominent by traction upon the palate. Pollock's method of relieving muscular tension, however, is simpler and easier of execution than Fergusson's, quite as effective, and is usually followed at the present day.

The operation is usually performed as follows. The patient is placed upon a suitable table in a good light and the anæsthetic administered, the surgeon being to the right side. Some prefer, in order to avoid the embarrassment of hemorrhage, to have the patient's head hang extended over the edge of the table, that the blood may flow out of the mouth instead of backward; but the position is an awkward one, and scarcely necessary, as a capable assistant can keep the mouth sufficiently clear of blood, by sponges in long holders, to avoid danger or much inconvenience. Smith's gag, or Wood's modification of it, should then be applied, having been previously fitted to the mouth, as shown in Fig. 16.

FIG. 16.



The patient's face is now turned towards the operator, who proceeds to freshen the margins of the fissure. The uvula being seized with toothed forceps and the velum put upon the stretch, it is transfixed above the forceps by a sharp, narrow, long-handled knife sufficiently far from the edge to give as large a raw surface as practicable, and the incision made downward to the tip; then the blade is reversed and the incision carried up to and around the upper angle. The opposite side is similarly treated, thus removing a continuous strip of tissue. If the operator be ambidextrous, this, as well as other steps of the operation, will be made much more easy. When the cleft extends quite up to the hard palate, it is essential to detach the soft parts from it for a short distance with a periosteal elevator; otherwise a sinus is very likely to result at the upper angle.

The next step, the passing of the sutures, is the most difficult and tedious, and many plans have been suggested and practised to lessen its embarrassments. The choice of these plans depends in a great measure upon the suture-material to be used, of which there is quite a variety. Wire, which is preferred by many, is most expeditiously inserted by the hollow needle with reel of Mr. T. Smith, the only objection to it being the difficulty of making punctures exactly opposite and at equal distances from the margins, —an objection which is common to all methods of passing the needle directly from side to side. The wire may be also drawn through by a thread loop previously placed, after the method of Avery.

As each suture, of which three or four are usually requisite, is passed, its ends should be lightly twisted and given to an assistant, to be held out of the way of the operator. When all are in position, the surgeon uses them for making traction upon the palate whilst he performs the next step, —that of section of the necessary muscles. As has been said, this is most conveniently done by Pollock's method.

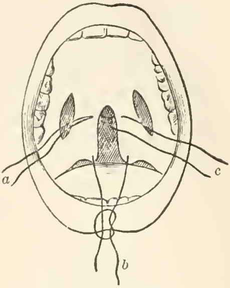
A narrow-bladed knife in a long handle is inserted through the palate to the inner side and a little in front of the hamular process, and the blade pushed backward with its cutting edge downward; now the handle is raised and an incision made downward in withdrawing the knife, thus severing the tensor and levator palati, without making an anterior wound much larger than the blade. The opposite side is treated in like manner,—when, if the muscles have been properly cut, the velum will be found less contractile and flaccid. One or both pillars of each side may thus be readily cut, if thought necessary, with scissors.

The wires are now twisted sufficiently tight to bring the sides nicely in apposition, and the ends cut off.

Silk sutures are used by many of the most skilful operators, and are, as a rule, to be preferred. Antiseptically prepared Chinese twist should be selected for the purpose, and the sutures are most satisfactorily inserted after Avery's plan. An ordinary nævus-needle answers very well for placing them, but two spirally-curved needles, right and left, are better. A single thread from one side of the cleft is drawn through the opposite side by a loop, as shown at *a* and *c*, Fig. 17.

This method of passing sutures in cavities is now too common to need description in detail. For securing the sutures the slip-knot of Sir William Fergusson (*b*, Fig. 17) is preferable to any other, as it is less likely to be tied too tightly. One additional knot over the loop shown in the figure is all that is required. In all other respects the operation is the same as that with wire.

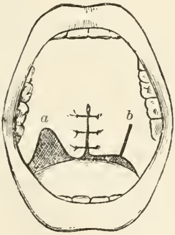
FIG. 17.



Some surgeons prefer to perform myotomy a day or two before closing the fissure, and others as the first step immediately preceding the paring; but the order given above appears to be the most satisfactory. Silkworm-gut makes an excellent suture, and horse-hair and chromicized catgut are also used.

While the methods of passing the sutures above given are considered the best, a dexterous operator may succeed perfectly in the operation with the ordinary curved surgical needle in a holder inserted from side to side in the usual way; or two such needles may be used, one to each end of the thread, both introduced from behind forward; but these methods are not to be recommended. In some cases it may be well to follow the plan recommended by Mr. Bryant, of placing the sutures as the first step, then pare the margins, tie the sutures, and paralyze the muscles by making an incision through the palate upon each side, as shown in Fig. 18 (*a* and *b*).

FIG. 18.



The sutures are generally withdrawn from the fourth to the eighth day, and until union is assured and firm the child should be kept upon liquid diet and talking interdicted.

Uranoplasty is the name given to the operation for closing cleft of the hard palate. It may be performed at the time of the staphylorraphy when the fissure involves both palates, or it may be deferred until after success upon the velum has been assured: when the bony portion alone is affected, it should be operated upon preferably at the age of three or four years.

Dieffenbach was the first to establish the practicability and success of the operation by the use of double flaps, and this method was improved by Von Langenbeck by including in them the periosteum.

The gag is applied, and the operation begun by freshening the edges of the cleft, which should be thoroughly done. An incision is then made down to the bone near the gum, extending from the last molar tooth as far forward as necessary, always well in front of the anterior angle of the fissure. With a periosteal elevator, curved or bent at right angles, the periosteum and soft parts are detached together from the bone by working carefully from without inward.

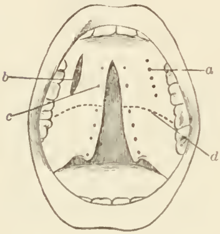
It is difficult to accomplish perfectly, but it is important that the flap should contain as much as possible of the periosteum, to avoid sloughing, and for the reproduction of a more or less perfect plate of bone. The separation should be done with deliberation and great care, or the tissues will be bruised or lacerated to such an extent as to hazard success.

The knife and elevator should not be carried so far backward and forward as to endanger the palatine vessels. When this step is completed on both sides, the flaps hang from the palate attached only at their extremities.

Hemorrhage is often quite profuse, but is usually controlled by pressure. The sutures are now inserted, beginning in front: the method of applying them and the choice of material do not differ from those in the previous procedure.

A portion of the bony palate is left exposed to the outer side of the flaps, which heal by granulation. The after-treatment is the same as after the operation upon the velum.

FIG. 19.



In some cases, and especially in those of failure, more or less complete, following the above method, Fergusson's osteoplastic operation may be tried. Holes are drilled with an awl along the margins of the cleft for the sutures, as shown at *c*, Fig. 19, an incision (*b*) made down to the bone midway between the free border and the gum, and the hard palate cut through with a chisel: to facilitate the chiselling, holes (*a*) may be previously drilled in the line of the incision.

The edges of the cleft being pared, the loosened portions of the palate are drawn inward, and the sutures tied in the middle line.

Fergusson in his later operations dispensed with the sutures, and substituted plugging the lateral openings with lint to retain the parts in apposition; but it is questionable whether this should be considered an improvement. The flaps should fall together readily without traction.

INJURIES AND DISEASES OF THE JAWS.

By J. EWING MEARS, M.D.

INJURIES.

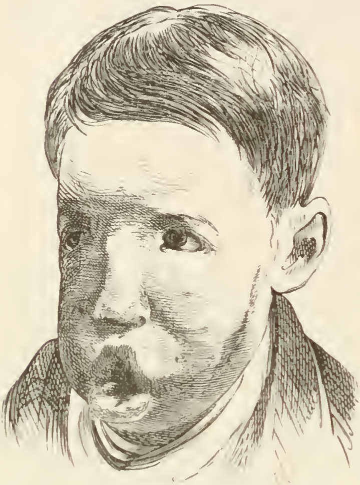
FRACTURES.

Superior Maxilla.—The firm position occupied by the superior maxillæ, between the bones of the head and the remaining bones of the face, supported on the sides by the zygomatic arches through the strong attachment of the malar bones, and behind by the pterygoid processes of the sphenoid bone through the tuberosities of the palate-bones, renders simple, uncomplicated fracture in other portions than the alveolar processes a rare accident. When fracture is sustained it is the result of great violence applied directly, as the kick of a horse, the blow of a bludgeon, or a crushing force, such as is exerted when the face is caught between the bumpers of railroad-cars in motion. Gunshot and shell wounds of the jaws, especially the latter, are attended, as a rule, with greater laceration and comminution of the bones than fractures due to other causes.

Of all portions of the bone the alveolar process is the most frequent site of fracture, owing in part to its more exposed position and to the presence of the teeth in the alveoli, the extraction of which is frequently a cause of fracture. Children share with adults the general immunity from fracture of the superior maxilla which the latter possess, and further enjoy a freedom from accidents of this character owing to the absence of exposure to causes incident to adult life. When fracture occurs in that portion of the bone in which the development of the teeth takes place, the process of second dentition may be very seriously interfered with, if not entirely destroyed.

The *symptoms* of fracture of the superior maxilla are, as a rule, well marked, including pain, loss of function, and, when the injury is the result of great violence, mobility and deformity. Crepitus cannot, as a rule, be elicited unless separation from the adjacent bones has occurred or comminution of the fragments is present. Wiseman, quoted by Hamilton ("Fractures and Dislocations"), reports the case of "a child with his whole upper jaw forced in by the kick of a horse, detaching the vertical plate and lateral masses of the ethmoid and forcing the palate-bone against the back of the pharynx." Hamilton (*loc. cit.*) also records a case by Harris, of New York, in which a child two years old, having fallen from a height of fifty feet upon the pavement, was found to have a diastasis of both the superior maxillæ and palate-bones, the separation being sufficient to admit the little finger, and extending from the position of the central incisors in the alveolar border to the soft palate. Prof. Agnew describes, in his Surgery, the case of a lad who had been caught between the bumpers of two railroad-cars, separating the whole face from the head and comminuting the upper and lower jaws (Fig. 1).

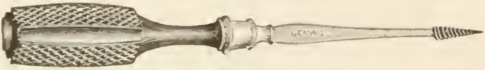
FIG. 1.



Appearance after recovery from an extensive fracture of the bones of the face accompanied by several wounds of the soft parts.

The principal indications for *treatment* are adjustment of the fractured and displaced bones, so as to avoid permanent deformity, and the preservation and replacement of all fragments, as it has been observed that the tendency to repair is very great in the upper jaw. Readjustment of the fragments may be accomplished by manipulation, aided in difficult cases by strong hooks and screw elevators (Fig. 2). The displaced fragments may be brought forward by placing the hooks behind the horizontal plate of the palate and making traction combined with manipulation. The screw elevator may be employed by making an incision over the malar bone and introducing the instrument into the bone, and in this manner the depressed bones may be elevated. Owing to the absence of action of muscular structures upon the bone, the fragments after adjustment are, as a rule, readily retained in place. Retention is effected by the closure of the jaws and by the application of Rhea Barton's or Gibson's bandage to maintain apposition (Figs. 3 and 4). Efforts may be made, by the applica-

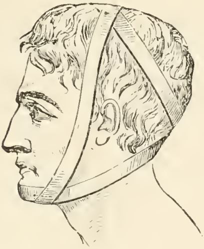
FIG. 2.



Screw elevator.

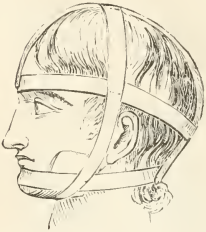
tion of compresses and adhesive strips, to retain the fragments in position. When the alveolar process is the seat of fracture, the interdental splint, made of hard rubber (Figs. 5 and 6), should be employed, closure of the jaws being maintained by the bandage. The patient should be kept upon liquid diet until repair is effected.

Fig. 3.



Barton's bandage for fracture of the inferior maxilla.

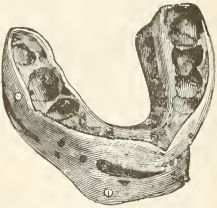
Fig. 4.



Gibson's bandage for fracture of the lower jaw.

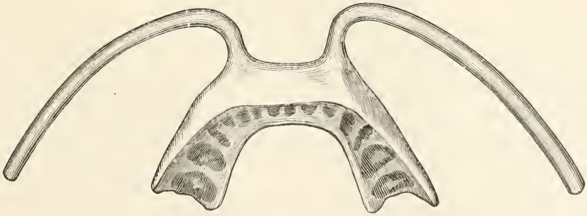
Inferior Maxilla.—The exemption from fracture which is observed to exist with regard to the superior maxilla in children equally attends the lower jaw. Of one hundred and fifty-eight cases of fracture of this bone recorded by Prof. Agnew as having been admitted into the Pennsylvania Hospital, five only were under ten years of age. The record of cases does not sustain the statement generally accepted, that the most frequent site of fracture in children is at the symphysis before union has taken place. Hamilton (*loc. cit.*) in a collection of eight cases of this variety of fracture found but one in a patient under ten years of age. As in adults, the body of the bone most frequently suffers fracture, rarely the angle or the processes.

Fig. 5.



Gunning's interdental splint.

Fig. 6.

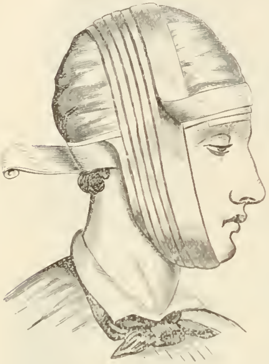


Kingsley's interdental splint.

The symptoms of fracture of the body of the lower jaw are those which attend fractures generally, and are usually so well marked as to make the diagnosis easy. In fractures of the ramus or processes the symptoms are frequently obscure, rendering detection of the character of the injury difficult.

The treatment of fractures of the body consists in effecting reduction by manipulation, and retaining the parts in apposition by means of external or interdental splints, and a bandage to maintain closure of the jaws. In cases where apposition cannot be maintained by the ordinary dressings the fragments may be held together by strong wire introduced through openings made with the bone-drill. Wiring the teeth is objectionable, and, as a rule, ineffectual.

Fig. 7.



The crossed of the angle of the jaw for fracture through the neck of the condyle. The turns should exactly overlap one another.

In fractures of the ramus or processes the fragments should be adjusted by pressure and counter-pressure, the fingers being placed in the pharynx, if necessary, to accomplish the object effectually. Difficulty is frequently experienced in maintaining the parts in apposition. The dressing usually applied consists of a compress over the parts and the bandage known as the "crossed of the angle of the jaw" (Fig. 7). The replacement of dislocated teeth in fractures of the jaws is not of the same importance in children, except where the permanent set is involved, as in adults. When not in the line of fracture, it is proper to replace them.

DISLOCATIONS

AT THE TEMPORO-MAXILLARY ARTICULATION.

The fixed position of the superior maxillæ renders dislocation without fracture or displacement of adjacent bones impossible. The movable character of the temporo-maxillary articulation admits of dislocation due to external violence or muscular contraction, the latter being the principal cause.

Study of recorded cases of dislocation at this joint and clinical experience have shown that in children this accident is of extreme rarity. Of nine hundred and twelve dislocations admitted to the Pennsylvania Hospital, as recorded by Prof. Agnew, only nine involved this joint, and none of these occurred in persons under twenty-five years of age. Sir Astley Cooper reports, as the result of his extended experience, but a single instance, which occurred in a boy who had an apple thrust into his mouth while at play. The peculiar conformation of the bone, owing to the absence of the full development of the alveolar border, and the obtuse character of the angle at which the rami are attached to the body, are given as reasons by which the non-occurrence of dislocation in the young is explained. Confirmation of this view is found in the fact that dislocation at the temporo-maxillary articulation is of rare occurrence in the aged, in whom the alveolar border is largely absent and the body and rami are joined at an obtuse angle. An explanation more satisfactory may be found in the absence of exposure to the direct causes of dislocation on the part of the young and of those far advanced in years.

The symptoms of dislocation are characteristic, and vary in degree according to the variety of displacement. When bilateral and complete, the mouth is widely open, the lower jaw is immovable, the chin protrudes, and saliva dribbles from the mouth. In normal position the condyles may be distinguished. In dislocation the finger may be passed into the depression formed by the glenoid fossæ of the temporal bone. Pain is usually present.

Reduction is effected by raising the chin, so as to unlock the condyles and place them upon the articular eminences in such position that the muscular tension is relaxed and they can be readily pushed into the glenoid fossæ. The manipulation is accomplished by introducing the thumbs, well protected, into the mouth, placing them over the molars on each side, and grasping the base with the fingers. In this manner the jaw can be firmly held, and the movements necessary to restore the condyles to their normal positions executed.

INFLAMMATORY AFFECTIONS.

ALVEOLAR ABSCESS.

The term alveolar abscess has been limited to the collection of pus formed at the apex of the root of a tooth. Abscesses forming at any other part of the alveolar cavity, as the result of injury or disease, should, according to Black,[1] if designated as alveolar abscesses, be accompanied by some qualifying adjective expressing the nature of the cause, as traumatic alveolar abscess, etc.

Cause.—Limiting the application of the term to inflammation at the apex of the root of the tooth, the cause may be always regarded as taking origin in the pulp of the tooth, and as being conveyed through the canal of the root to the tissues surrounding its apex. Exposure of the pulp in a carious tooth leads readily to inflammation of the structures at the apex.

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[1] System of Dentistry, vol. i.

In children, by reason of the relation of the deciduous to the forming permanent teeth, suppuration in the alveolus may seriously involve the latter.

Stages.—Clinically the stages may be divided into those which characterize the inflammatory process, leading to resolution or suppuration.

Symptoms.—In the first or beginning stage of the inflammation the symptoms are quite marked. Of these symptoms pain is the most prominent. Beginning usually as a dull pain in the affected tooth, it soon becomes extreme. It is in this stage due to the pressure exerted in the confined space at the root of the tooth by the engorged blood-vessels. The swelling of the structures is sometimes sufficient to cause a slight protrusion of the tooth from the alveolus. Pressure upon the tooth in the effort to force it back into the alveolus causes an increase of pain. The tissues of the gum soon become painful and discolored, passing, as the inflammation advances, from a deep red to a purplish hue. Heat over the site of the affected tooth and swelling of the side of the face are also symptoms which accompany this stage.

In the second stage, when plastic exudation has occurred, an abatement of the symptoms takes place. The deposit of plastic matter at the apex of the root increases somewhat the extrusion of the tooth, and this condition may be accompanied by an increase of pain. In other respects the symptoms are less pronounced.

If in the third stage resolution takes place, the symptoms gradually disappear. If, on the other hand, suppuration supervenes, an aggravation of all the symptoms occurs. This event may be ushered in with a rigor, followed by elevation of temperature and increase in pulse-rate. The tension produced by the pus confined in the small space at the apex of the root augments greatly the pain. Absorption of the wall of the alveolus at the apex takes place rapidly, and the pus makes an effort to escape by the way of least resistance, reaching the surface, as a rule, on the buccal or cheek side of the mouth-cavity. When the pus leaves the cavity of the alveolus, it may dissect a route for itself beneath the periosteum and appear at the surface at the junction of the gum with the tooth, or, perforating the periosteum, infiltrate the overlying tissues and finally seek an exit over the root of the tooth or escape at the margin of the gum along the peridental membrane. The escape of the pus from the alveolar cavity is attended by a diminution of the pain and a marked swelling of the structures of the face, effecting in some cases closure of the eye upon the affected side, with closure to a greater or less extent of the jaw.

In the form of abscess in which the pus in its escape from the alveolus passes beneath the periosteum, detachment of this membrane occurs to a greater or less extent and necrosis is liable to result. The pointing of the pus varies according to the teeth affected and the resistance offered. In the upper jaw abscesses connected with the anterior teeth occasionally point over the hard palate, dissecting off the periosteum in some instances to a great extent, or into the nasal cavities. Abscesses involving the posterior teeth may open upon the face, just in front of the anterior border of the masseter muscle or into the maxillary sinus.

In those connected with the teeth of the lower jaw the pus by gravity may descend to the base of the jaw, and when the abscess is accompanied by necrosis it may point in the middle of the neck or at the upper border of the clavicle.

Diagnosis.—The diagnosis of alveolar abscess is, as a rule, not difficult, the symptoms being well marked and the presence of a carious tooth indicating the cause. It is to be distinguished from periostitis in the early stages by the localized character of the symptoms and the presence of an affected tooth.

Treatment.—In the early stages efforts should be directed to the promotion of resolution and the prevention of suppuration. Treatment of the affected tooth is of the greatest importance, and should receive the earliest attention. This should consist in such treatment as will provide for the preservation of the tooth, or its extraction if its condition is such as to forbid this. Removal of the diseased pulp and clearing of the canals of the roots, with the use of antiseptic agents and temporary fillings, will constitute the treatment in the effort to subdue the inflammation and to save the tooth. When the carious condition is such as to render the tooth useless its extraction will at once determine the relief of the inflammatory action. General and local measures may be resorted to in the early stages to assist in the accomplishment of resolution, the most efficient of the latter being section by the bistoury of the inflamed tissues overlying the affected tooth, and the application of absorbents. The employment of hot fomentations over the face should be *scrupulously avoided*, as such applications invite the pointing of the pus to the surface.

When suppuration has occurred and the pus has not escaped from the alveolus, an effort should be made, by clearing the pulp-cavity and the canals of the root, to secure its removal through the tooth. If an abscess-cavity has formed outside of the alveolus its early evacuation into the mouth is imperatively demanded, in order to prevent pointing upon the surface, with the resulting formation of a scar.

The importance in children of retaining as long as possible the deciduous teeth should not be lost sight of in the treatment of alveolar abscess. The absorption of the roots of the primary teeth which occurs may afford greater facility in the treatment of such abscesses through the tooth, permitting thus the retention of the affected organ.

PERIOSTITIS.

Inflammation of the periosteum of the jaws may occur in the acute or chronic form, being due in the first to the irritation of carious teeth, to the eruption of permanent teeth, to injury, to a specific poison, such as occurs in the eruptive diseases, to ptyalism, or to phosphorus vapor. In tuberculous children periostitis may occur without any apparent cause.

A syphilitic taint of the system is usually the cause of the chronic form of periostitis. The extension of the periosteum into the alveoli, forming the peridental membrane, leads to an involvement of the teeth in acute periostitis and to the presence of symptoms relating to them.

Symptoms.—The symptoms of periostitis are pain of a diffused character, heat in the part affected, and swelling of the face, with more or less general constitutional disturbance. The involvement of the peridental membrane in the inflammatory process causes protrusion of the teeth from the alveoli, with great pain on pressure.

Diagnosis.—The diagnosis is made by careful inquiry into the cause and examination of the teeth and tissues overlying the jaws. The rapid occurrence of suppuration leading to necrosis renders the formation of an early and correct diagnosis important.

Treatment.—Prompt depletion by general and local means is the treatment which should be adopted in acute periostitis of the jaws of the ordinary form. Saline cathartics should be freely administered, with sedatives to allay pain. The removal of blood by leeches and by incisions of the periosteum should be practised, and energetic measures taken to prevent suppuration.

In syphilitic periostitis, which is attended by less urgent symptoms, iodide of potassium should be given until the pain and swelling subside. Caution is to be especially observed with regard to the extraction of teeth in this form of periostitis, as such operations are very liable to excite acute symptoms followed by suppuration and necrosis. The dull pain felt leads the patient to apply to the dentist, who may under a mistaken diagnosis perform extraction. In such cases the history should be carefully ascertained, and the patient placed for a sufficient time upon specific remedies before tooth-extraction is attempted.

NECROSIS.

As has been stated in the remarks upon periostitis, this condition precedes necrosis, and hence the great importance of adopting such measures in treatment as will check the inflammatory action and prevent the occurrence of suppuration.

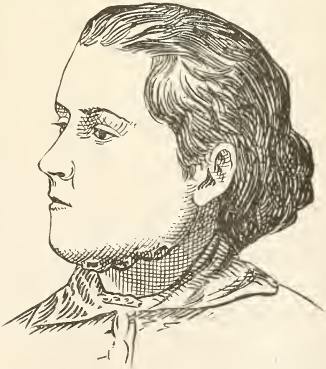
Of the various forms of necrosis the variety most liable to occur in children is that induced by the specific poison of the exanthemata. Formerly, in this country especially, ptyalism was often a cause of necrosis in children, owing to the injudicious use of mercury; to-day it is of rare occurrence. Syphilitic necrosis is found in adults, as a rule, and phosphorus-necrosis occurs, according to my experience, most frequently in young persons between fourteen and twenty years of age, the time of life at which they engage as employees in match-factories. Other causes may produce necrosis, as the irritation of carious teeth, the irruption of the permanent teeth, mechanical injury, as produced sometimes in the extraction of teeth, and ulcerative affections, as cancrum oris and scorbutus.

Where ordinary periostitis follows the irritation caused by carious teeth or the infliction of injury and leads to necrosis, it is the result of the inflammation of structures in a normal state. In exanthematous necrosis the specific poison of the disease exerts its influence upon the periosteum, inducing inflammation and necrosis. In syphilitic necrosis the specific poison attacks the osseous structure, with its covering the periosteum. Ptyalism excites irritation of the salivary glands and inflammation of the structures covering the alveolar processes and inner surface of the cheeks, leading to necrosis. I have endeavored to show elsewhere[1] that in phosphorus-necrosis a chronic toxic condition is established which awaits some exciting cause to make itself manifest: this is usually found in the efforts made to extract carious teeth, or in the irritation caused by collections of tartar, or by the use of pins, which are used by employees in their work, in picking the teeth.

The symptoms of necrosis are so well marked that no difficulty should be experienced in making a diagnosis. A careful inquiry into the history of the case will lead to the determination of the exciting cause and the variety present.

The prophylaxis consists in the prompt treatment of the preceding periostitis. When suppuration is established, measures should be taken to limit the necrotic action. This is best accomplished by drainage and evacuation of collections of pus, and by thorough cleansing of the mouth and abscess-cavities with antiseptic solutions. Experience has shown that early operative interference is harmful and should not be practised, the resulting injury inflicted causing almost invariably an extension of the inflammation. In children during the presence of the temporary teeth the expectant plan of treatment should always be employed, sufficient time being afforded to permit complete detachment of the sequestrum, so that the permanent teeth if unaffected by the disease may not be injured and their evolution and growth not be interfered with. The pus which is formed may point upon the face (Fig. 8), and efforts, by incision and drainage of pus-cavities, should be made to cause evacuation into the mouth. When these efforts are unavailing and abscesses form upon the surface, they should be incised and drained, in order that the resulting cicatrix shall produce as little deformity as possible. The administration of

Fig. 8.



Showing abscesses at the base of the lower jaw in a case of phosphorus-necrosis.

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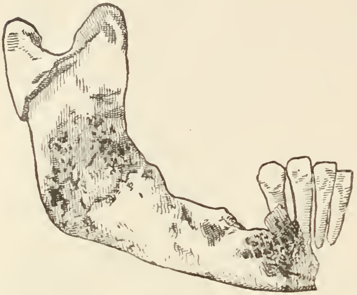
[1] Transactions of the American Surgical Association, vol. iii.

tonics and nutrients, good diet, with exercise in the open air, will contribute to the general well-being of the patients and increase their powers of resistance to the harmful effects of the suppurative action.

Fig. 9.



One-half of a lower jaw removed by incisions within the mouth.

Removal of the sequestrum should always be effected through the mouth, avoiding thus an external incision (Fig. 9). In the lower jaw the plates of new-formed osseous matter should be carefully separated from the sequestrum, and the latter lifted from its position. If the sequestrum is entirely free it can be liberated by the bone-pliers or chain saw, using the instruments so as to inflict as little injury as possible.

The reproduction of bone occurs in necrosis affecting the lower jaw, affording, when it has not been interfered with by too early and injudicious efforts at removal of the sequestrum, a substantial support for an artificial denture. In a number of cases of phosphorus-necrosis in which I have removed one-half, two-thirds, or the whole of the lower jaw, artificial dentures have been adopted and worn with comfort by the patient.

Reproduction of bone does not occur in necrosis of the upper jaw, the cavity left after the removal of the sequestrum being filled partially or completely by fibrous tissue.

DISEASES OF THE ANTRUM.

CYSTS.

The imperfect development of the antrum in the young subject renders the occurrence of cystic disease (hydrops antri) or suppuration in the cavity very rare. The importance of the study of these affections relates largely to their differentiation from tumor of the antrum and of the upper jaw, as cases are recorded in which extirpation of the jaw has been performed through an error in diagnosis, cystic disease having been mistaken for tumor.

Formerly supposed to be a collection of fluid in the antrum as the result of occlusion of the opening between the nasal cavity and the latter, later investigations have shown that hydrops antri is due to the cystic degeneration of the glandular follicles of the mucous lining membrane, the gradual dilatation of one or more cysts expanding the walls of the cavity and forming a swelling which may simulate a solid growth. The slow growth, the absence of pain, and the resiliency of the anterior wall, yielding with a crackling noise under pressure, constitute the principal symptoms of this affection, which should be carefully studied in making a diagnosis. If any doubt should exist, puncture through the anterior wall within the mouth will disclose the character of the contents.

The treatment consists in gaining access to the cavity either by a puncture of the wall above the alveolar border or, preferably, by extraction of the first molar tooth, and thorough curetting of the interior, in order to disintegrate the degenerated follicles and permit their removal by the instrument, assisted by repeated douching of the cavity with hot water and subsequently with antiseptic solutions. Drainage may be effected by a small canula or tube, the mouth of which should be stopped with a pledget of cotton to prevent the intrusion of particles of food, which, undergoing decomposition, might lead to inflammation. The application of tincture of iodine or other stimulating agents, care being taken to afford free escape, may assist in the curative process.

ABSCESS.

Abscess or suppuration in the antrum may be caused by blows over the part, or by injury to the alveolar process, through which the cavity is opened, or, as is most frequently the case, it may result from the carious condition of one or more of the teeth in relation with the cavity, as the second bicuspid, or the first and second molars. Inflammation beginning in these teeth may be conveyed by continuity or contiguity of structure and involve the lining membrane of the cavity.

The symptoms of this affection differ from those of cystic disease in their more acute character: the onset is more rapid, and the symptoms of inflammation are very marked: heat, pain, increased on pressure, redness, and swelling are prominent. The pressure of a carious bicuspid or molar tooth in a state of inflammation will assist in arriving at a correct diagnosis with regard to the cause and nature of the disease. The resilient condition of the anterior wall gives information as to the presence of a fluid in the antrum, as in cystic disease.

The existence of pus in the sinus having been determined, its evacuation, followed by douching with hot water and antiseptic solutions, should be accomplished. Access can be had to the cavity by extraction of the carious tooth which is usually present as the cause of the suppuration in the antrum. The opening between the alveolus and antrum can be enlarged by the passage of a small trocar with canula, care being taken to guard with the index finger the extent to which the instrument enters the cavity. The canula may be left in position, for the purposes of drainage and injections. If suppuration has followed removal of the process, leaving a large opening, the interior may be packed with antiseptic gauze, and douched with antiseptic solutions when the packing is changed.

CYSTIC DISEASES.

CYSTS CONNECTED WITH THE TEETH.

Two forms of cysts are described which are connected with the teeth,—first, those found in connection with completely developed teeth, and, second, those in which the teeth are in process of development and have not irrupted.

The first are of inflammatory origin, and are found at the apex of the roots of carious teeth. They are formed from the peridental membrane, which is detached from the root of the tooth and forms a sac for the fluids which are produced. The roots of the teeth are usually in a state of disease and have undergone absorption. In children, when inflammation attacks the roots of the deciduous teeth absorption of the roots occurs, and cysts are formed which frequently interfere with the treatment of the inflammation through the tooth. These cysts vary in size, being in some instances very small and in others quite large. Heath[1] quotes a case reported by Fisher, in which a cyst connected with the apex of the posterior molar tooth and filling the whole antrum grew from the periosteum of the apex of the root of the tooth.

The treatment of these cysts consists in the extraction of the affected tooth, by which the cyst remaining attached is removed. If it does not come away with the tooth it should be removed by a small curette: if left, suppuration continues. When very large, and cavities in the alveolar process have formed, they should be removed by incision through the bone.

Dentigerous cysts, or those originating in connection with undeveloped teeth, are formed, as described by Mr. Tomes (quoted by Heath, *loc. cit.*), "by the detachment of the investing soft tissue from the enamel surface of the tooth by a small quantity of transparent fluid which not uncommonly collects in the interval so formed. This fluid ordinarily is discharged when the tooth is cut, but when from some cause the eruption of the tooth is prevented it increases in quantity, gradually distending the surrounding tissues in the form of a cyst." In the lower jaw these cysts sometimes acquire a large size, and in some instances the jaw has been excised under the belief that a tumor existed. An important diagnostic point relates to the absence of permanent teeth which should, at the period of life when the examination is made, be present. When sufficient expansion has occurred to render the bony walls thin, a crackle will be heard on pressure. Further information may be obtained in cases of doubt by puncture with a small trocar, within the mouth, by which means the existence of fluid will be determined.

The treatment of the dentigerous cysts consists in incision of the bony wall within the cavity of the mouth, evacuation of the contents, removal of the undeveloped tooth, which may be found projecting into the cavity or lying loose on its floor, and finally approximation of the sides, thus securing obliteration of the cavity by crushing in the walls.

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[1] Injuries and Diseases of the Jaw.

CYSTS OF THE LOWER JAW.

According to Heath (*loc. cit.*), this form of cyst originates in the cancellated tissue of the bone, which is lined with the endosteum, in this respect differing from cysts of the antrum, which arise in the mucous follicles of the lining membrane which have undergone cystic degeneration. He thinks it not unreasonable to attribute the origin of the cysts to some irritation connected with the roots of the teeth, a cancellus expanding and producing gradual absorption and obliteration of those adjacent until a cyst of considerable size is formed. The cysts may be unilocular or multilocular, and contain usually a viscid fluid. By the pressure exerted, they cause expansion and, in some instances, great distortion of the jaw.

The same methods of examination should be employed in determining their character as in the other forms of cysts described above. They are to be distinguished from cystic osteo-sarcoma by the absence of any tendency to infiltrate the adjacent tissues. They are also of slow growth and painless.

Cysts of the lower jaw should be treated by free incision within the mouth, removal of the contents, and obliteration of the cavity by crushing in the walls. In addition to these measures, Dr. Mason Warren, of Boston, advocated the use of injection to maintain a sufficient degree of irritation to favor the deposition of new bone.

TUMORS OF THE JAWS.

SUPERIOR MAXILLA.

Tumors of this bone take origin most frequently in the cavity of the antrum or in the alveolar border, rarely from the facial, zygomatic surfaces or palatine process. With regard to the relative frequency of their occurrence, my experience accords with that expressed by Prof. Gross,[1] that sarcoma embraces rather more than one-third and carcinoma less than one-third of all neoplasms of the superior maxilla, cystoma, osteoma, fibroma, and chondroma occurring in the order named. In children my experience leads me to believe that sarcomas are of the most frequent occurrence, and that of the different forms the spindle-celled predominates largely.

Fibromata.—Fibromas appear in the antrum, but most frequently upon the facial surface or alveolar process, taking origin from the periosteum covering the surface or from its prolongation into the alveoli, where it forms the lining membrane,—the peridental membrane. Growths developed in connection with this membrane are termed fibrous epulis. Fibromas may contain pure fibrous tissue, nodules of cartilage, spiculæ of bone, or cysts. When developed in connection with the periosteum covering the facial surface, they are usually attached by a broad base. In a tumor of this nature removed by the author its base covered the entire facial surface, and it was encased in a shell of bone, with osseous spiculæ interspersed throughout the growth. Fibromas possess all the characteristic features of benign growths, and are differentiated from sarcomatous and carcinomatous formations by slow growth, painlessness, circumscribed character, absence of glandular involvement, and lack of tendency to return after extirpation. The growths may be removed by cutting around the base with a strong knife or chisel and prying them off from their bed, little hemorrhage accompanying the operation.

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[1] System of Surgery, 1883.

Fibrous epulis takes origin from the peri-alveolar membrane, is usually attached by a pedicle, is small, with smooth and regular surface, of dense consistence, sometimes bleeding freely from contact of food, and liable to undergo ulceration.

While benign in its character, permanent relief can be obtained only by removal of the alveolus from which the growth springs, or of the alveolar border if more than one alveolus is involved, so as to embrace in the excision the limits of the disease. Many cases have presented themselves at the clinic of the author in which attempts have been made by dentists to remove these growths by ligature or section of the pedicle. In every case these efforts have been futile, and in some of them extension of the disease has occurred, requiring greater loss of structure for radical cure.

Cartilaginous Tumors form very rarely in connection with the jaws. A few cases are reported by Prof. Gross (*loc. cit.*) as being developed between the bone and periosteum on the facial surface and nasal process. One case of pure chondroma of the antrum he states is recorded by Rindfleisch, and one of sarcomatous chondroma by Mr. Stanley in a lad sixteen years of age. They resemble other benign growths in their general features. A characteristic feature is their appearance at an early age. They sometimes grow to a large size. Removal effects permanent cure.

Osseous Tumors.—These tumors occur in middle-aged and old people, and are the result of external violence or of syphilitic disease. They may form upon any part of the jaw, usually the facial surface or alveolar process, portions most exposed to external violence. Instances of osseous tumors developing within the antrum are recorded. All forms are amenable to treatment by extirpation, the chisel and gouge or saw being required to obtain separation from the bone.

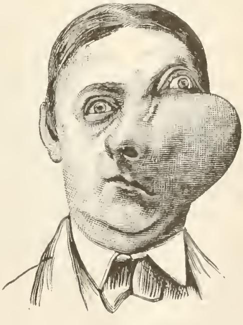
Sarcomatous Tumors.—As stated above, of all tumors of the jaw the sarcomata are the most frequent and occur principally in the young. They present several varieties. The *periosteal*, composed of spindle cells, and in some instances of small round cells, when its consistence is less firm, is found in connection with the facial surface and within the antrum.

The myeloid sarcoma appears at an early age, and attacks, as a rule, the alveolar process at the canine and bicuspid portion. It consists of giant cells largely embedded in spindle-celled tissue, grows rapidly to a large size, and invades in some instances the cavities of the antrum, the mouth, and the nose. In some cases, also, it is very vascular, the vessels being so distended as to give the growth a distinct pulsation. *Sarcomatous epulis,* like the fibrous variety, takes origin from the periosteal lining membrane of the alveoli, and consists of spindle cells. Another form is described, originating from the medulla-cells of the Haversian canals, which is composed largely of giant cells. The symptoms of the two forms distinguished as the periosteal and the myeloid differ somewhat, the former presenting, in the slow growth and firm consistence, the characteristics of the fibroma, while the latter resemble in the more rapid growth and softer structure, especially when ulceration has taken place, the medullary carcinoma.

The treatment consists in free excision of the parts involved, with the bone, when the growth is small, and, when large, of the entire jaw. The remarks made with regard to excision of the process in fibrous epulis may be applied in stronger manner with regard to the treatment of sarcomatous epulis. Exsection of the portion of the process by pliers or saw, extending beyond the limits of the disease, is imperative. Any operation which does not include this is worse than useless: it is positively harmful.

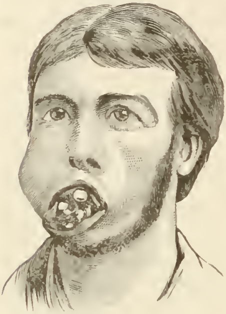
Carcinomatous Tumors.—This form of neoplasm presents itself as encephaloid, epithelioma, and, rarely, scirrhus. The two first mentioned resemble each other very closely anatomically and clinically. They occur usually after the twentieth year, being most common after the fortieth year. When situated in the body of the bone the disease begins usually in glands of the mucous lining membrane of the antrum, and the growth, increasing rapidly in size, in some cases pushes through the walls of the cavity and invades the cavities of the mouth, nose, and eye, the fauces, and the frontal sinuses, and even penetrates the cranial cavity through its floor. Soon ulceration attacks the overlying mucous membrane and integument, and a fungous mass is formed which pours out a fetid discharge containing, in most cases, blood. In the case represented in Fig. 10, the tumor, originating in the cavity of the antrum, had invaded the mouth, nose, orbit, fauces, and frontal sinuses, and had caused absorption of the base of the cranium. At the time of the operation for its removal these cavities were exposed. Glandular involvement soon occurs, and the patient dies from pain and exhaustion. The rapid growth, infiltration of the surrounding structures, painful character, and tendency to undergo ulceration, distinguish these growths and make the diagnosis quite easy.

Fig. 10.



Carcinomatous epulis appears in the glands of the mucous membrane overlying the gums, and gradually involves the bone. It presents a cauliflower-like surface, is very painful, exhibits a great tendency to undergo ulceration and to bleed, and gives rise to a fetid, sanious discharge. Its history and appearance are sufficiently characteristic to enable it to be easily distinguished from fibrous and sarcomatous epulides. On section and microscopic examination of these forms of tumors they will be found to consist of squamous and cylindriform epithelium contained in a soft alveolar basis-structure. The osseous structure involved is soft, porous, and disorganized: large blood-vessels with very brittle walls permeate the substance of the tumor. If removed at an early period, when small and the bone is not involved, the prognosis of carcinomatous epulis may be regarded as favorable. When the growth has progressed so as seriously to implicate the bone, excision of the jaw should be performed, as in the case represented in Fig. 11, in which the tumor was melanotic in character and had invaded the entire bone.

FIG. 11.



The treatment of carcinomatous affections of the upper jaw by operative procedure is limited to the early period of their development, before the extensive infiltration of the surrounding structures. If delayed beyond this time, the inability of the surgeon to secure complete extirpation renders a speedy return of the growth inevitable. In some cases of extensive involvement, when deglutition or respiration is interfered with by the encroachment of the growth upon the fauces, operation is indicated to relieve the symptoms present and to permit painless death.

INFERIOR MAXILLA.

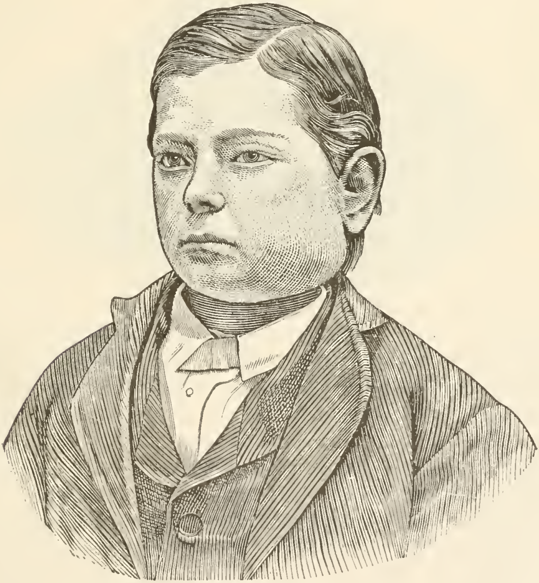
The various forms of tumors of the jaws which are described above as occurring in connection with the upper jaw occur more frequently in connection with the lower jaw. They take origin from different points,—the surface, the interior, or the alveolar border,—and grow in some instances to great size.

Fibrous, cartilaginous, and osseous tumors of the lower jaw arise, as a rule, from the external surface of the body of the bone, and do not differ in any respect from those which are found in connection with the upper jaw. They present similar symptoms, and may be readily distinguished from sarcomatous and carcinomatous formations by the symptoms which characterize benign growths. Fibromas and chondromas occur most frequently in the young, and may acquire large dimensions, while the osseous, which may be spongy or ivory-like in character, attack the middle-aged or the old, and do not grow to great size. Myxomatous tumors occur rarely, and then in connection with the interior of the bone developing from the medulla. They possess the features of growths arising from the interior of the bone.

The treatment of the tumors above mentioned is the same as that advised in regard to those of the upper jaw, complete extirpation of the growth resulting in permanent relief.

Sarcomata.—Sarcomatous tumors of the lower jaw resemble in every respect those of the upper jaw, whether central or peripheral. The myeloid variety appears as a central growth, and, as a rule, in the young about the period of second dentition. Heath (*loc. cit.*) records a number of cases of this form of sarcoma in the young, one, rare in character, in a boy seven and a half years old, which involved both sides of the jaw and appeared at the age of eighteen months, growing very gradually and without pain. The projecting portions of the growths were sawn off and the interior of the bone gouged away, with permanent relief.

FIG. 12.



Figs. 12 and 13 exhibit the appearance presented in a patient of the author by a peripheral sarcoma of the left side of the lower jaw in a lad twelve years of age, before and after the operation for its removal. The tumor appeared after the receipt of a severe blow upon the part, and developed in three and a half months to the size represented in the drawing.

Owing to a condition of closure of the jaws which was present, the growth was removed by an external incision. On making the dissection the masseter muscle at its point of insertion was found incorporated in the tumor. The microscopical appearances of the tumor were those of a small round-celled structure with a delicate, finely-granular, occasionally fibrillated, intercellular tissue. A point of great interest was revealed in the presence of some muscular fibres which showed intramuscular sarcoma. The report of the microscopic characters of the tumor, by the late Dr. Bertolet, states "that these intramuscular cells, still enclosed by the sarcolemma, have a round, occasionally an oval shape, a large nucleus which is readily stained with carmine and surrounded with a thin layer of protoplasm. In size, these cells equal those of the primary growth. . . . The sarcous elements seem to passively disappear under the pressure of the newly-formed cells without previously becoming granular or fatty. The transverse and longitudinal structures of the muscular fibres finally disappear entirely, and naught but cylinders filled with round cells, each separated by a fibrillated intercellular tissue, remain,—*i.e.*, the muscular substance has been transformed into young cellular connective tissue, and sarcomatous cells have been differentiated." Up to this date, now sixteen years, recurrence of the growth has not taken place.

Fig. 13.

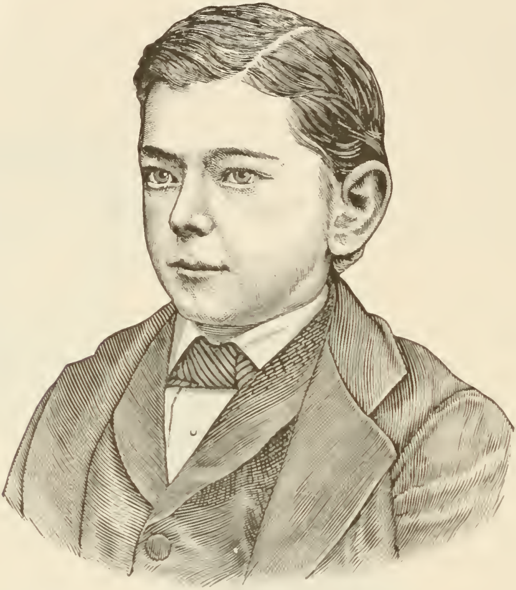
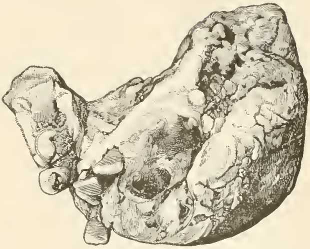


Fig. 14 shows sarcomatous epulis in an advanced stage occurring in a boy ten years of age. In this case I removed the greater portion of the body of the jaw, and thus far there has been no recurrence. The tendency manifested by sarcomatous epulis to return after operation demands that in every case free excision of the parts involved should be made, even if necessary to the complete removal of a portion of the bone, as in the case reported.

Fig. 14.



Carcinomatous Tumors.—These growths appear more frequently in connection with the lower jaw than with the upper, the variety most frequently present being the encephaloid or medullary. Scirrhus is rare. They are characterized by the certainty with which they return after operation, without regard to early or late operative interference. Heath (*loc. cit.*) records a case of medullary disease of the lower jaw in a child aged five, in which he performed two operations, removing one-half of the jaw in the first operation, and on return of the disease six weeks later one-half of the remaining portion of the jaw. Notwithstanding the prompt surgical measures taken, the child perished in a little more than six months after the first appearance of the disease. As carcinomatous affections of the lower jaw are peculiar to old persons, the case above noted is a rare example of the disease in so young a subject.

EXCISION OF THE UPPER AND LOWER JAWS.

Operations for the removal of morbid growths of the upper and lower jaws should, whenever it is practicable, be performed without external incision. Small tumors involving the alveolar processes may be readily and thoroughly removed by operation within the mouth. Non-malignant growths occupying the facial surface of the upper jaw or the external surface of the body of the lower jaw may be, in some cases, attacked and successfully extirpated from within the mouth. The removal of the sequestra after necrosis, with disarticulation at the temporo-maxillary joint if necessary, can be effected by internal incision. Carcinomatous growths if subjected to operation demand free excision, which in most cases can be accomplished only by removal of the entire bone, which must be done by external incision.

In removal of the upper jaw the incision which exposes the bone fully, produces least deformity, and divides the facial artery and nerve at points where the branches are small, is one which is curved and which begins near the angle of the mouth, passes along the ala of the nose to near the inner angle of the eye, and then curves out to a point over the centre of the malar bone or to its zygomatic process.

In excision of the lower jaw the incision should begin in front of the lobe of the ear, and be continued over the angle to the base, and along the base to the symphysis, and thence upward to the border of the lip, if one-half is to be removed. If this latter portion of the incision can be avoided, it is better to do so, as it leaves a cicatrix which is quite perceptible. If the entire jaw is to be removed, the incision along the base may be continued to the lobe of the ear of the opposite side.

CLOSURE OF THE JAWS.

Permanent occlusion of the jaws may be due to the presence of dense cicatricial tissue in the buccal spaces, to the formation of plates of osseous material between the alveolar processes, or to bony ankylosis at the temporo-maxillary articulation. With the exception of two cases, all that have come under my observation and care have occurred in children between the ages of six and eleven, although operations for relief have not in some cases been performed until adult life.

Of the cases in which closure was due to cicatricial tissue, ptyalism, and gangrenous stomatitis following eruptive fevers, were the causes, while gunshot wounds, fracture, injuries to the joint, and inflammation were the causes of synostosis or bony ankylosis. One or both sides of the buccal cavities may be affected in cicatricial closure, or one or both of the temporo-maxillary joints in ankylosis.

Closure taking place before the period of second dentition interferes very much with the development and irruption of the permanent teeth. In two cases in which occlusion occurred in early life, periods of seventeen and twenty-seven years elapsed before permanent opening was accomplished, at which time eighteen and twenty-two teeth, embedded in the processes and projecting horizontally and at various angles from them, were removed. In other cases sufficient mobility existed to permit of the irruption of the permanent teeth with less irregularity. In two instances the upper and lower central incisors had been worn away by the continuous rubbing of masses of food against them, through which efforts the nutriment was extracted. In other cases a slight space existed between the upper and the lower teeth, through which liquid and very soft food was pushed into the cavity of the mouth. Notwithstanding the defective means of taking food which existed in all cases, nutrition seemed to be quite well maintained. In patients of adult life the existence of the disability caused more or less depression of spirits, through which their general health was affected. Death from asphyxia was narrowly averted in one case, a boy twelve years of age, who was seized with a violent attack of vomiting caused by an over-indulgence in pie, masses of which he had forced into his mouth and swallowed.

In all cases in which closure occurred during childhood and a long period elapsed before operations for its relief were performed, imperfect development of the jaws was observed. In one instance the patient, although over thirty years of age, had a decided "baby" face, the features being those of a child, while the stature and physical development were those of a tall and rather large woman. Soon after the operation which opened the mouth and gave motion to the lower jaw the expression changed. A young man who had suffered from occlusion since his eleventh year of age and who obtained permanent relief at twenty-eight was unable to cultivate a beard until after the operation which afforded free movement to the lower jaw.

Frequently much suffering occurs from carious teeth which can neither be treated nor extracted, owing to the firm jaw-closure.

Treatment.—The treatment of jaw-closure may be considered under three heads: first, that which relates to immobility due to the formation of cicatricial tissue; second, that which relates to immobility due to ankylosis of the temporo-maxillary articulation; and third, that which relates to immobility due to the presence of osseous plates between the alveolar processes.

Until a recent period the treatment of all these varieties has been very unsatisfactory. Systematic treatises on surgery, both in this country and abroad, either entirely omitted mention of the affection and its treatment or contained but a meagre reference.

Dr. Valentine Mott in 1847, in his "Concluding Remarks" in the American edition of Velpeau's "Operative Surgery," stated that immobility of the lower jaw had never, so far as he knew, formed a distinct chapter in any systematic work on surgery. He further stated that he had had during the previous thirty years a number of cases and had treated them successfully.

He described three different forms, dependent upon different causes: first, closure due to unyielding cicatrices, the cause of which cicatrices he does not mention; second, a preternatural rigidity or dynamic contraction of the muscles, the result of the violent action of mercury upon the mouth and adjacent parts; and third, osseous union by means of a bony plate which extended from the coronoid process to the superior maxillary bone. With regard to this form he states that, if such a condition of things could be known at the time, probably some sort of operation might be devised to sever or exsect this bony communication and thereby at once open the jaws.

In his treatment of cicatricial closure he relied upon incisions and the use of an instrument constructed upon the screw-and-lever principle. After division of the cicatricial bands and the use of the lever he was enabled to open the mouth, and this apparently was regarded as a cure, as no report is given of the condition months later. So unsatisfactory has this method proved in the hands of surgeons since the date of Dr. Mott's operations that Prof. Esmarch, then of the University of Kiel, and Prof. Rizzoli, of Bologna, both abandoned interference with the cicatrices, and advocated and practised the formation of a false joint in front of their position. This plan was feasible only when but one side was affected, and it also gave as its result the use of but one-half of the jaw, with accompanying deformity.

In the first case of cicatricial contraction (Fig. 15) which came under my care I divided the inodular tissue a number of times and applied an instrument for a long time, without attaining any permanent satisfactory result. In studying the case it occurred to me that the reunion of the divided tissue could be prevented by first forming behind it a track, by means of a large ligature which should remain loosely in position until the inner surface of the canal thus formed should heal. I accordingly made use of this method,—partially only, however, by reason of the indisposition of the patient to submit to further treatment. Imperfect as the effort was, I succeeded in effecting permanent opening to the extent seen in Fig. 16,—three-quarters of an inch. In this case cicatrices existed upon both sides and were very dense and strong.

Fig. 15.

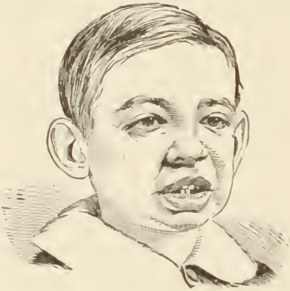
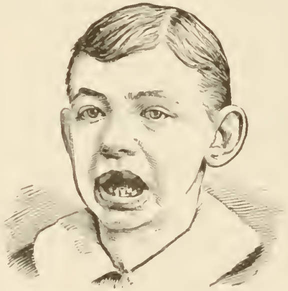


Fig. 16.



In passing the ligature a needle with a handle and with the eye at the point is used: this is introduced at the anterior margin of the cicatrix and carried between the integument and the cicatrix to its posterior margin, where it emerges and the ligature is seized with the forceps and the needle withdrawn. Where the bands are so dense as to obliterate the buccal spaces they should be divided within the mouth by the probe-pointed bistoury, and the mouth opened sufficiently wide to permit the passage of the armed needle. In two cases which I have operated upon by this method since the above case, I have obtained permanent separation in one case of one inch, and in the second case of one inch and three-quarters, the report of this extent of separation in the last case being made at the expiration of one year following the operation.

Mr. Heath (*loc. cit.*), in speaking of closure due to ankylosis at the temporo-maxillary articulation, states that the treatment of cases of the kind is "eminently unsatisfactory." When the ankylosis is complete he advises division of the ramus, as Dieffenbach had proposed for cicatricial contraction, as easier and safer than excision of the joint. Division of the ramus could be effected within the mouth by dissecting up the mucous membrane and masseter muscle and then introducing a narrow saw or strong bone-forceps with which the section could be made. The difficulty, and in some cases the impossibility, of preventing reunion of the divided ramus after simple section has prevented the adoption of this plan of operation. Excision of the joint by external incision has been employed in a number of cases with success,—with greatest success in those in which the osseous deposit has been slight. The joint may be exposed by a horizontal incision carried along the base of the zygoma or by one which is vertical over the joint.

The failures in many cases to accomplish complete and satisfactory separation of the jaws by simple section of the ramus or excision of the condyle induced the author to attempt the formation of a permanent false joint by section of the ramus of the bone and removal of both condyloid and coronoid processes. In the later operations I have accomplished this by dissection, within the mouth, of the mucous membrane and masseter muscle overlying the ramus, and section of the bone by an Adams saw. The upper fragment is seized with the lion-jawed forceps and turned out, the temporal and external pterygoid muscles being divided by the probe-pointed bistoury. If the bone yields at the neck of the condyle the articulatory surface may be removed by the chisel if sufficient space has not been acquired. The masseter muscle may also be divided if it is found necessary owing to its rigid character. Figs. 17 and 18 show the condition before and after an operation performed in the manner above described for the relief of bony ankylosis following necrosis after an attack of scarlet fever. When closure is due to the existence of osseous plates, relief can be afforded by introducing a narrow saw between the teeth and dividing the plates.

FIG. 17.

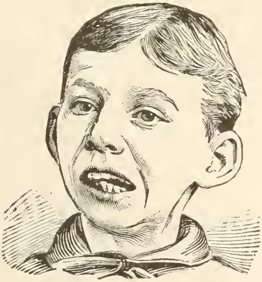
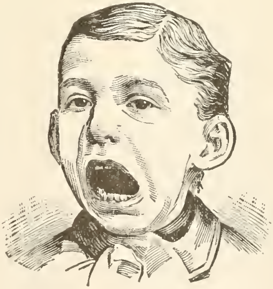


FIG. 18.



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